CONDITIONED REFLEXES
AND
NEURON ORGANIZATION

By

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DEDICATED TO
I. P. PAVLOV AND C. S. SHERRINGTON
IN THE HOPE THAT THIS WORK
WILL DO SOMETHING TO BRIDGE THE GULF
BETWEEN THEIR RESPECTIVE
ACHIEVEMENTS

We thus, from the biological standpoint, see the cerebrum, and especially the cerebral cortex, as the latest and highest expression of a nervous mechanism which may be described as the organ of, and for, the adaptation of nervous reactions.. . It is then around the cerebrum, its physiological and psychological attributes, that the main interest of biology must ultimately turn.

(C. S. SHERRINGTON, *The Integrative Action of the Nervous System*)

On the whole, looking back upon this new field of physiological research I find it full of fascination, especially since it satisfies two of the fundamental cravings of the human intellect - striving to realize ever new and new truths, and to protest against the pretension of finality in truth we have already gained. In this domain there will for long remain an immense breadth of uncharted ocean compared with the small patches of the known.

(I. P. PAVLOV, *Conditioned Reflexes*)

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FOREWORD

It is now forty years since Sherrington's outstanding monograph, *The Integrative Action of the Nervous System,* was published. To a large extent this book laid down the roads of the further development of physiology of the nervous system, and outlined a programme of experimental research for many years after

True, the experimental material with which Sherrington operated at that time was comparatively small, and a number of his generalizations proved to be erroneous. But if Sherrington's book has played such an enormous part in the development of neurophysiology, and is even to-day of great actual, and not only historical, importance, it is because the author has put forward a certain *general conception* of the functioning of the central nervous system, which still to-day we have to regard as sound and fruitful. Broadly speaking, this conception consists in basing the functioning of the central nervous system on its neuron organization, in laying down that isolated reflex arcs are only an abstraction and that the nervous system acts as a single integrated whole, that the organism's reactions to stimuli falling on it are always multi-effector, using an enormous number of final common paths in various ways and in various combinations. Working on these principles, Sherrington carried out a brilliant analysis of the interaction of reflexes, indicating the manner in which they participate in the co-ordination of the organism's activity. Obviously it is difficult to foresee how the Sherrington conception will be regarded by later generations of physiologists, what part of it will be rejected, and what additions we shall have to make in order to allow for newly obtained facts, but it must be objectively admitted that at the present time the physiology of the central nervous system is developing to a large extent under the guidance of this particular conception, and that for the time being we do not see any likelihood of its possibilities being exhausted.

If we attempt to trace in general outline the further development of investigation into the problems raised in Sherrington's monograph, we have to state the following. The problems of integrative action continued to be studied in Sherrington's

laboratory and also in other physiological laboratories, covering both the reflex activity of the spinal cord and that of the subcortical ganglia. But gradually problems in this field have been exhausted, and the centre of gravity of research work has shifted more and more in the direction of problems concerning the intimate nature of the intercentral transmission, the mechanism of the processes of excitation and inhibition, the exact structure of particular reflex arcs, etc. As experimental technique grew more perfect, and especially as the apparatus for recording action potentials improved, analysis of the processes occurring in the central nervous system became more subtle and exact, and so the state of our present knowledge (despite the circumstance that certain important problems are still unsolved) is incomparably more precise than the data available to Sherrington forty years ago.

So, while investigations into the 'microstructure' of the activity of the nervous system have recently made enormous advances, in the field of 'macrostructure' of the functioning of the nervous system, i.e. in that of integrative action, we have made incomparably less progress from the position which was reached in Sherrington's first monograph. I suppose that this is chiefly due to the fact that investigations concerning this subject were performed mainly on decorticated and spinal preparations, and they did not include the main integrating organ, the cerebral cortex.

As is well known, an extensive investigation of the cerebral activity was undertaken by Pavlov, who studied it over many years with the aid of the method of conditioned reflexes which he had created. But the theory of the nervous processes which Pavlov developed on the basis of his own and his collaborators' experimental work is entirely different from Sherrington's conception, and in consequence the work of the Pavlov school is not a genuine extension of Sherrington's work into the field of higher nervous activity, but constitutes a separate and independent line of inquiry.

The task which has been undertaken in this book consists in an attempt to extend the Sherrington conception of the functioning of the nervous system to the field of higher nervous activity. This has been done on the basis of the enormous experimental material collected by the Pavlov school over almost forty

years of research work All this factual material has been isolated from the theoretical conceptions in which it has been entangled, and reinterpreted and reorganized from a new viewpoint.

Basing our analysis of the higher nervous activity upon the Sherringtonian principles, in other words, accepting that the nervous impulse is that form of nervous process which causes the excitation in nerve cells and carries it from one neuron to another, we do not in the least forget, or even disregard, the great body of experimental facts accumulated during recent years concerning the spontaneous rhythmic activity of the brain, which facts suggest that there may be other mechanisms of propagation of the nervous processes. We think, however, that, whatever their interest and importance, these facts are yet not sufficient to serve as a foundation of a new theory of the nervous processes, and it is not even excluded that they may be explainable in terms of the Sherringtonian conception. On the other hand, in our analysis of the body of facts dealt with in the present book we are not confronted with any necessity to go beyond the 'classic' principles of the nervous function and accept any additional assumptions which may be, but also may not be, true This explains why in the present book the vast consequences of modern electro-encephalographic study have not been utilized and taken into consideration. This does not mean, however, that we deny the possibility that new mechanisms of nervous function may eventually emerge from that study, and that they may throw light on many facts hitherto entirely unintelligible, perhaps even improving upon or replacing our present explanations.

It must be pointed out that the new approach to the study of the higher nervous activity confronts us with a large number of new questions, to some of which we do not find answers in the existing experimental data. And so certain of the problems surveyed could be solved only hypothetically on the basis of inadequate or indirect factual evidence, while others have only been posed, without any attempt at their solution. So the question is bound to arise, whether a book of this kind ought not to be postponed until all the more important consequences arising from the new approach to the field discussed are experimentally worked over and elucidated.

The following considerations disposed me against such an attitude The field of experimental investigation which is opened to the experimenter by this proposed approach to the physiology of higher nervous activity is really very extensive, and greatly exceeds the possibilities of the physiological laboratory at my disposal in the Nencki Institute of Experimental Biology, especially as it was completely destroyed during the war and has had to be reconstructed from the beginning On the other hand, it seems to me that an adequate and exact interpretation of the experimental material already collected by Pavlov's collaborators from the point of view of the general laws governing the functioning of the nervous system, making that material comprehensible and clear to modern neurophysiology, is of importance in itself, and should be made as soon as possible.

Obviously, any attempt to reinterpret the data of the physiology of higher nervous activity requires as corollary the demonstration that the theory which Pavlov formulated and acted upon is incorrect. That involved presenting an outline of the Pavlov theory and subjecting it to exhaustive criticism.

This exposition of the general principles of the Pavlov theory differs in two respects from the synthetic expositions which Pavlov himself wrote during the last years of his life.* On the one hand, it presents not only cortical mechanisms with which the Pavlov theory operates, but also those experimental facts on which this theory has been based, facts which were almost completely omitted in Pavlov's last published works. On the other hand, as our book is confined to consideration of problems of the normal physiology of higher nervous activity, problems of pathophysiology are hardly touched upon.

As to criticism of the Pavlov theory, this has here been confined to purely physiological questions, and does not touch upon the philosophical problems which are often widely discussed in association with Pavlov's conception

In this connexion I think it desirable to state the following:

For many years I took Pavlov as my teacher. Both when working in Leningrad under his direct guidance and later in Poland at the Nencki Institute of Experimental Biology, I was

* Vide Pavlov, I. P , *Lectures on Conditioned Reflexes*, vol II. London, 1941.

always greatly influenced by his scientific ideas and governed by his conceptions. However, as the years passed, I became more and more convinced that Pavlov's theory was not correct, as it could not be reconciled with the evidence of general physiology of the central nervous system, nor was it adequate as a working hypothesis for explaining experimental facts. For that matter, as is well known, Pavlov himself realized that his theory contained many defects and contradictions, but he ascribed them to 'the devilish complexity of the subject', as he used to say, and not to the unsound prerequisites on which he had chosen to base his analysis.

Thus, I consider that the time is ripe to submit Pavlov's doctrine to a thorough and detailed criticism and to make an attempt to replace it by another which would better fit the experimental facts and which would be compatible with the general principles of neurophysiology It hardly needs stressing that no reflexion is cast thereby upon Pavlov's scientific work, it would rather seem to be the best tribute that could be paid to his memory and achievements to attempt to establish adequate foundations for a further development of the branch of science which he has created and to which he devoted the greater part of his life, instead of leaving it fettered to an erroneous theory.

An undoubted defect of this book is that it was written during a period when, owing to the war, I was almost entirely cut off from western literature, and my own notes were destroyed. I have attempted to fill some of the hiatuses (though certainly not all) during my stay of some months in London. And so I hope I may be pardoned by anyone who finds views similar to his adduced in this book without acknowledgement, and also by anyone who finds views contrary to his own stated without any explanatory criticism of his viewpoint.

A few words on the literature quoted. Whenever describing various facts in the field of higher nervous activity, I refer to the original papers only when there have been few papers on the given subject or when a certain work is regarded as the classic for a given problem. But I do not provide any references when discussing generally known facts which every investigator in this field knows through his own experimental practice. As the

papers of Pavlov's collaborators are in general inaccessible to the British and American reader, in all cases where Pavlov has quoted and discussed the given paper in his books I cite the corresponding page of their English editions.* Thus the reader will be able to acquaint himself directly with the problems which are of greater interest to him, or to check and supplement the factual evidence I have given. In regard to the English terminology of the Pavlov theory, I have in general kept strictly to that adopted by Anrep and Gannt in their excellent translations. Only here and there has it seemed useful to make insignificant changes in their terminology.

I cannot end without expressing my deep gratitude to all those who have helped me in the publication of this book. In particular I am deeply obliged to Professor C. H. Waddington, General Editor of the Cambridge Biological Studies, for all the interest he has shown in and all the support he has given to the publication of this book. I would like, too, to thank Professor E. G. T. Liddell, Dr L. Lubinska, Professor J. H. Woodger and Professor J. Z. Young very sincerely for reading the manuscript and making most valuable suggestions and corrections. And I have to put on record my thanks to the translator for his able and ready co-operation.

I would like to add that it was through the assistance of the Polish Ministry of Education that I was able to visit this country in order to complete this book and to prepare for its publication, and that all the expenses connected with the translation were covered by the Nencki Institute of Experimental Biology. I also owe a debt of gratitude to the British Council for their valuable help extended to me in respect of my visit to England.

J.K.

LONDON,

November, 1946

* Pavlov, I. P., *Conditioned Reflexes, An Investigation of the Physiological Activity of the Cerebral Cortex*, translated and edited by G. V. Anrep, Oxford University Press, Humphrey Milford, 1927. *Lectures on Conditioned Reflexes*, translated and edited by W. Horsley Gannt, Lawrence and Wishart Ltd. London, vol. I, 1928; vol. II, 1941 In further references to these books we shall call the first simply *Conditioned Reflexes* and the second *Lectures*.

CHAPTER I

Conditioned reflexes and the physiology of the central nervous system

1

There is one aspect of research in the physiology of higher nervous activity which cannot but be regarded as unsatisfactory by many workers in this field. It is this. Whereas in Russia and later in the Soviet Union the science of conditioned reflexes developed very rapidly, and even took precedence over other departments of physiology, attracting many workers, it has aroused comparatively little interest among physiologists in other countries. It is true that Pavlov has long enjoyed great authority among scientists, and the fame of his investigations during both the first and the second, main, period of his activity, has spread all over the world. None the less, whereas his discoveries in the field of digestive functions have become a permanent acquisition of physiology, and it is impossible to deny or even ignore them, the study of conditioned reflexes, to which he devoted far more years of strenuous work, has not yet become part of the body and blood of physiology, and is still situated somewhere on the periphery of this science. To use an expression which is familiar to all who were among his disciples, we would say that the science of conditioned reflexes still remains *concentrated* in the centre of its origin, and irradiates far too feebly into other centres of physiological thought.

Of course conditioned reflexes are well known, and a great deal is written and said about them not only among scientists but also among the laity. But, unfortunately, all the hubbub which, justly or unjustly, has arisen around Pavlov's theory is little concerned with its substance, but is chiefly interested in its philosophical bases, the problem of the relations between psychology and physiology, the possibility of a physiological approach to the study of behaviour, etc. Whatever its philosophical importance, such discussion does not contribute anything positive to the physiology of higher nervous activity,

any more than discussion of the existence of infinitesimal magnitudes advances mathematical analysis.

However, it has to be pointed out that of recent times the physiology of higher nervous activity has penetrated deeply into related fields, such as psychology, pedagogics, neuropathology and psychiatry, and that it is now playing a large and positive part in these fields. As is well known, conditioned reflexes are of particular importance in the American behaviourist school of psychology. But if the manner in which behaviourism exploits the science of conditioned reflexes is analysed more closely, it will be found that it consists chiefly in utilizing the basic and most elementary concepts of this field, i.e. the Pavlovian *nomenclature*, for the denomination of particular phenomena (e.g. the experimental extinction, differentiation, generalization, etc.) without getting down to their physiological content and physiological mechanism. And if at times behaviourist psychology borrows anything more from the physiology of the higher nervous activity and makes use of such concepts as irradiation or induction, it does so purely as a borrower, utilizing these concepts in the same way as physiology makes use of concepts of physics and chemistry. How little the achievements of the Pavlov school have been taken into account, even by those investigators who have been most directly concerned with the study of conditioned reflexes, may be judged, for example, from the chapter on the conditioned reflex written by Dr H. S. Liddell of Cornell University in Fulton's famous monograph, *Physiology of the Nervous System* (second edition, 1943). Stating that the influence of Pavlov's theory on neurophysiology 'has been almost nil' and that 'it is at present of historical interest only', Liddell simply disregards the huge body of experimental evidence which forms the bases of the theory and which must find explanation in some way or other. It is easy to decry Pavlov's theory, but then one is obliged either to explain all its basic facts in some other way or to admit that they are at present unexplainable. Liddell, however, takes the inadmissible course of drawing a veil of silence over most of the facts and deals only with the most elementary phenomena in the field. One regrets to have to say that many American authors behave in exactly the same way.

So we are faced with a strange and abnormal situation in the modern physiology of the nervous system. More or less at the same time, at the turn of the nineteenth century, two related branches of physiological science began to develop systematically and almost independently of each other. There was the physiology of the lower nervous activity, and especially the spinal cord, dealt with in the work of Sherrington and his school, and the physiology of the higher nervous activity, in the Pavlovian teaching on conditioned reflexes. Both these disciplines developed rapidly and fruitfully, but whereas the first has spread all over the world and is now studied in many scientific centres and schools, and its achievements have become a generally recognized acquisition of physiological knowledge, the other has so far hardly passed outside the bounds of the school which created it, has not been enriched by the results achieved by other schools, and still to-day remains isolated from other, even closely related, departments of physiology.

How has this situation come about? Of course it is partly due to the fact that the majority of works on the physiology of the higher nervous activity are published in Russian, a language comparatively little known in other countries. But it is easy to show that this explanation is quite inadequate. For almost all Pavlov's own work has been translated into many other languages, and is available to all the world. Besides, the very fact that the science of conditioned reflexes is well known to American behaviourists, i.e. to scientists who in general do not know Russian, shows that the chief obstacle is not that of the language difficulty, but that there are other factors hindering the transmission of knowledge in this field to physiologists outside the Soviet Union. We shall consider these other factors in a moment.

2

Any physiologist who has studied the field of conditioned reflexes, and who also knows the present state of physiology of the lower nervous activity, cannot but be greatly surprised at the extent to which these two related departments of science use quite different languages, not in the linguistic, but in the conceptual sense. It is true that there are certain terms which

are common to both departments. Both groups of physiologists use the terms excitation and inhibition, irradiation, induction, and facilitation. But the properties ascribed to the phenomena denoted by these terms are so different that the identity of language rather makes the situation worse, since it gives an impression of homogeneity where it really does not exist. And so, an English or American neurophysiologist who reads Pavlov's surveys of the functioning of the cerebral cortex,* not being acquainted with all the experimental evidence from which the laws there formulated are inferred, is often at a loss to know what they mean and whence they originate. Thus, when he reads that the process of excitation or inhibition first irradiates over the cerebral cortex from the cortical point in which it arose, and then concentrates back, or that a concentrated cortical process induces the inverse process on its periphery, that a weak excitation (or inhibition) irradiates over the cortex, that one of moderate strength is concentrated, but again that a strong one irradiates, or that a strong excitation of the cortical cells brings about a protective or top inhibition, etc., then, as he reads all this he can only be puzzled at the extent to which the laws of functioning of the cerebral cortex differ from those governing the lower nervous activity.

The question arises whether the differences between the cerebral cortex and other parts of the nervous system really are so great. As for the morphology, we know that the accumulation of neurons is greater in the cerebral cortex than elsewhere, their topography and relation to the white matter are different, but the plan of structure and general pattern of neuronic connexions are the same throughout the nervous system. As for their respective functions, we have to note one property which is peculiar to the cortex, or at least is displayed by it to an incomparably higher degree than by lower parts of the nervous system. It consists, broadly speaking, in the following: while a stimulus impinging on the spinal cord gives rise to a more or less prolonged cycle of events after which the organ returns to its previous state, a stimulus reaching the cerebral cortex evokes,

* E.g., 'A brief outline of the higher nervous activity' (*Psychologies of 1930*), Pavlov, *Lectures*, vol. II, p. 44. 'Physiology of the higher nervous activity' (read before the 14th International Physiological Congress, Rome, 1932), *Lectures*, vol. II, p. 86.

besides disturbances analogous to that occurring in the cord, another kind of change which leaves permanent traces in the cortex. In other words, while the property generally called excitability is an attribute of the entire nervous system, the cerebral cortex (and probably to some extent the subcortical ganglia also) exhibits an additional property which may be designated *plasticity* (vide Chapter v).

Taking the foregoing into account, we have to conclude that, when undertaking investigation of the function of the cerebral cortex, we should be prepared for the fact that this organ does possess certain specific properties distinguishing it from other parts of the nervous system, and that the language in which its activity has to be described needs to be enriched by certain new terms. But we cannot expect this language to be fundamentally different from that of the physiology of the spinal cord. And yet the situation at present is precisely that the two languages are quite different, and there is not even a dictionary to explain the one in terms of the other.

It follows that a very important and pressing task confronting physiologists of the central nervous system is to bridge the gulf separating the two fields of research, and to unify the language in which each field is described. So long as this is not done the present abnormal situation, which is only a brake on the development of the science, will continue.

The problem that thus arises needs to be discussed unambiguously and without reservations. The basic properties which Pavlov ascribed to the processes of excitation and inhibition, the manner in which these processes are propagated, and their temporal evolution, all (as will be shown in later chapters) differ fundamentally from the properties of those processes that have been established in studying the functions of the lower parts of the nervous system. The interpretation and even the description of experimental facts given by Pavlov, and, after him, his disciples, in hundreds of papers on conditioned reflexes are fundamentally different from and often contradictory to the generally accepted interpretation of facts in the physiology of lower nervous functions We must conclude, therefore, that either the general physiology of the nervous system based on the experimental work of many research centres gives a wrong

interpretation of data and must be revised and enriched by the laws established in the study of conditioned reflexes,* or that Pavlov's interpretation is inadequate, and that the extensive experimental material collected by his school requires a different systematization and explanation.

In this rivalry of two physiological conceptions—the one created by Pavlov to explain the cortical phenomena, and the other built up on the basis of investigation of lower nervous activity—one should *a priori* admit the superiority of the latter. Indeed, since functional relations in the cerebral cortex are undoubtedly far more complex than those in the spinal cord, it seems sounder to utilize the latter in order to explain the mechanisms of cortical activity than vice versa, especially as, owing to modern experimental methods, the conditions of investigation of lower nervous activity have been perfected to the highest degree Besides, it must be remembered that the cerebral cortex, like the entire nervous system of the higher animals, possesses a neuronic structure, and any theory which does not take this fact into account (which is precisely the case with the Pavlov theory, as we shall show later) must eventually fail to stand.

After detailed analysis of the body of facts collected in this field by the Pavlov school, we came to the conclusion that these facts *can* be systematized and explained on the basis of the conceptions and data of neurophysiology, and that the system which can thus be developed, being a logical complement of the system of physiology of lower nervous activity, will make it possible to lead the science of conditioned reflexes out of the dead end in which it is at present. It is to the explanation and development of this new approach that this book is devoted.

* Pavlov himself frequently expressed this view, considering that the method of conditioned reflexes is more perfect than those adopted in investigation of the function of the spinal cord. By way of illustration of his opinion we quote the following passage: 'I think that the experiments on conditioned reflexes carried out on animals in normal conditions afford the formulation of general laws [concerning the whole of the central nervous system—J.K.] better and more precisely than could be done hitherto on the basis of data obtained from the lower parts of the nervous system and generally in acute experiments' (Lecture to the 14th International Physiological Congress, Rome, 1932).

Chapter II

An outline of Pavlov's theory of the activity of the cerebral cortex

1

As is well known, the fundamental concept of the physiology of the higher nervous activity is the *conditioned reflex*. In this science it plays a dual role On the one hand, it is regarded as the simplest functional mechanism specific to the highest levels of the nervous system (primarily to the cerebral cortex). On the other hand, in the work of the Pavlov school and of certain other investigators the conditioned reflex is treated as a *method* of investigating the activity of the cerebral cortex, i.e by means of the 'method of conditioned reflexes' the functional properties of this organ are made known

Without making any claim to exactitude, which would call for excessive comment, we may formulate the definition of a conditioned reflex as follows: a conditioned (or individually acquired) reflex arises when two stimuli, of which one evokes an inborn, permanent, or so-called unconditioned reaction of the organism, are applied in association a number of times, and it consists in the second of these stimuli beginning to evoke the same kind of reaction as that produced by the first stimulus. The stimulus which now gives rise to a reaction, which previously it did not evoke, becomes the *conditioned stimulus*, and the simultaneous application of both stimuli—unconditioned and conditioned—is called the reinforcement of the conditioned stimulus (or reflex) by an unconditioned stimulus (or reflex).

In experimental work on higher nervous activity, alimentary or acid conditioned reflexes (i.e reflexes which are reinforced by presentation of food or the introduction of acid into the animal's mouth) are generally used; the reaction observed is a flow of the saliva through the opening of the salivary duct transplanted to the outside of the cheek, and usually so-called indifferent stimuli (auditory, visual, tactile or olfactory) serve as conditioned stimuli. The conditioned and unconditioned

stimuli are usually applied in overlapping sequence, the first preceding the latter by ten to thirty seconds (the so-called isolated period of the conditioned stimulus). Such a procedure enables the observation of the conditioned response at each trial.

Pavlov was not particularly concerned with the mechanism of the actual formation of conditioned reflexes. He considered that this type of reflex arises by the establishment in the cerebral cortex of a connexion between the centre of the conditioned stimulus and the centre of the unconditioned stimulus (possessing its 'cortical representation'), but he was rather vague as to the nature of this connexion. And so the statements to be found in his writings, that 'every strongly excited centre in some manner attracts to itself every other weaker excitation reaching the system simultaneously, and thus the point of application of this excitation...becomes more or less firmly connected with that centre', or that a conditioned reflex is the result of 'the meeting of the waves irradiated from different points'* are rather vague hypotheses, and play no essential part in his theory.

The established conditioned reflex may be temporarily or permanently suppressed in two ways. The first consists in some extraneous stimulus, evoking its own unconditioned reaction, being applied together with the conditioned stimulus (or just preceding it), with the result that the conditioned reflex is inhibited to a greater or lesser degree. Pavlov called this inhibition, which is analogous to other similar phenomena in the nervous system, an *external inhibition* (also a passive or unconditioned inhibition), and the foreign stimulus exerting inhibitory effect he called the *external inhibitor*. The second method of suppressing a conditioned reflex consists in its ceasing to be reinforced by an unconditioned reflex. Here, at the very beginning of investigations into conditioned reflexes, a fact came under notice which played a great part in the development of the physiology of the higher nervous activity, and which must

* The first of these two quotations is taken from 'A brief outline of the higher nervous activity', an article published in *Psychologies of 1930* (*Lectures*, vol. II, p 47). The second is taken from a Lecture to the International Physiological Congress, Rome, 1932 (*Lectures*, vol. II, p. 87).

be regarded as one of the basic facts in this field. It was found that if the conditioned stimulus, in some way or another, ceases to be reinforced (see below), then, although it no longer produces the conditioned response, it by no means becomes what it had been before (e g. an indifferent stimulus), but acquires new, definite properties. During the initial stages of investigation into conditioned reflexes two such properties were observed. The first was that if, after presenting (best of all several times in succession) such an unreinforced and therefore inactive conditioned stimulus, another normal conditioned stimulus is given, the effect of the latter is weakened to a greater or lesser extent The second property then observed was that, if some not very strong foreign stimulus is applied simultaneously with an inactive conditioned stimulus, this latter is reactivated and gives rise to a conditioned reaction. These discoveries led Pavlov to the conclusion that non-reinforcement of a conditioned stimulus leads not to the destruction of the reflex, but to its inhibition, which manifests itself either in its being extended to other conditioned reflexes (the first of the above-mentioned properties, called *inhibitory after-effect*) or in the fact that under the influence of foreign stimuli it can be temporarily removed (the second of the above-mentioned properties, known as *disinhibition*).

The inhibition that arises in consequence of the non-reinforcement of a conditioned reflex Pavlov called an *internal* or *active inhibition*, since it arises in the actual arc of the conditioned reflex; and he called the stimulus which as the result of non-reinforcement had ceased to give a conditioned reaction the *internal inhibitor*, or *inhibitory* or *negative conditioned stimulus*. So it follows that besides the active or positive conditioned reflexes with a directly observable effect, there are also negative, or inhibitory conditioned reflexes, whose negative effect cannot be directly measured, though their existence can be inferred from their inhibitory influence upon other conditioned reflexes.

According to the manner in which the conditioned reflex concerned is not reinforced we can distinguish different varieties of internal inhibition.

The first and simplest variety occurs when the conditioned reflex ceases to be reinforced by the unconditioned stimulus.

This variety is called the *experimental extinction* of the conditioned reflex. Usually in experimental work use is made of so-called *acute* extinction, which consists in repeating the conditioned stimulus many times without reinforcement at brief (in terms of a few minutes) intervals. After a number of such repetitions, the conditioned reaction falls to zero. If the conditioned stimulus continues to be applied without reinforcement the state of inhibition is more and more 'deepened', which is manifested by an increasingly strong and more and more prolonged inhibitory after-effect (the so-called secondary extinction of other conditioned reflexes). If the extinguished stimulus is not repeated during a period of time, the conditioned reflex recovers spontaneously The longer the interval and the less deepened the inhibition, the more complete is the restoration of the positive conditioned reflex.

A second variety of internal inhibition arises in the following circumstances. It is known that if a conditioned reflex has been elaborated in response to a given stimulus, it is also evocable by other stimuli, *similar* to the original conditioned stimulus. It was established by experiments that similar stimuli are stimuli of the same quality but of different intensity, the same intermittent stimuli of different frequencies, tones of different pitch, tactile stimuli directed to different parts of the skin, etc. This phenomenon is called the *generalization* of the conditioned reflex. But if one of the similar stimuli is regularly reinforced by the unconditioned stimulus, while the other is never reinforced, then, after a certain period the second stimulus is *differentiated* from the first, and the positive conditioned reflex to it is transformed into an inhibitory reflex. This variety of internal inhibition is called *differentiation* or *differential inhibition*.

The so-called *conditioned inhibition* is a particular case of differentiation. It occurs when the given conditioned stimulus is reinforced, whereas the same stimulus, combined with an additional stimulus, is not reinforced.*

* The addition of an extraneous agent to the conditioned stimulus, of course, produces an external inhibition. But if the additional agent evokes only an orientation reaction, then on its repetition this reaction gradually fades, and with it also fades its inhibitory effect on the conditioned reflex.

After a time the combination of stimuli ceases to produce a conditioned reaction and becomes inhibitory. The additional stimulus, which has acquired the property of inhibiting the conditioned reaction to the stimulus with which it has been combined, is called the *conditioned inhibitor*. It must be mentioned that the conditioned inhibitor added to any other conditioned stimulus forms an inhibitory combination with it, without previous elaboration.

Finally, a third variety of internal inhibition occurs when the conditioned stimulus is followed by an unconditioned stimulus, not after a few seconds (as usually) but after two or three minutes from its inception. During the early applications of such combinations the conditioned response continues throughout the isolated period of the conditioned stimulus, but later this response gradually shifts more and more towards the moment when reinforcement occurs, and in its first phase the conditioned reflex is inhibited. This kind of internal inhibition is called the *inhibition of delay*.

2

The laws of conditioned reflex activity so far discussed have been of a purely descriptive character, and the terms introduced have served simply for the denomination and classification of the observed phenomena. Only in the next period of investigation into conditioned reflexes did problems arise which concern the central mechanism of these phenomena, in other words, which concern the mechanism of the activity of the cerebral cortex.

This period was opened with Krasnogorsky's experiments in 1910,* which were afterwards repeated in innumerable variations by various workers. The general scheme of the experiments we refer to was as follows. Conditioned reflexes to a number of similar stimuli (e.g. to the rhythmic touching of various places on a dog's skin, or to a number of different musical tones) were established, one of these stimuli was transformed into an in-

* Krasnogorsky, N I, Diss., Petersburg, 1911 (Pavlov, *Conditioned Reflexes*, pp. 153 et seq)

hibitor, and then the extent of the inhibitory after-effect upon active conditioned stimuli at various intervals after the application of the inhibitory stimulus was investigated. A dual kind of inhibition was made use of. Either one of the conditioned stimuli was acutely extinguished, and immediately after its extinction (more or less deep) the secondary extinction of other, similar conditioned stimuli was investigated; or one of the stimuli was differentiated from the others and, when the differential inhibition was adequately established, the inhibitory after-effect upon active conditioned stimuli applied after the inhibitory stimulus was investigated. By comparing the results of all the experiments of a given series, it was possible to determine the extent and the duration of the inhibitory effect exerted by the inhibitory stimulus on various active conditioned stimuli.*

The results obtained in the majority of experiments of this kind can be summarized as follows:

(1) The more the given conditioned stimulus resembles the stimulus originally inhibited (i.e. extinguished or differentiated), the more lasting is the inhibitory after-effect upon it.

(2) Given an equal interval after the application of the inhibitory stimulus, the inhibitory after-effect is the stronger, the more the conditioned stimulus resembles the inhibitory stimulus which it follows.

(3) The secondary inhibition of all conditioned stimuli applied after the inhibitory stimulus increases *gradually*, achieves its maximum after a dozen or so seconds, or rarely after a few minutes, and then gradually diminishes over the course of a few or a dozen or so minutes.

(4) The more times the inhibitory stimulus is repeated (i.e. the more the internal inhibition is deepened) the stronger and the more lasting is the inhibitory after-effect.

(5) The secondary inhibition of active conditioned stimuli

* The substantives 'inhibition', 'extinction', 'differentiation' and the corresponding participles are used in association both with the substantive 'reflex', and with the substantive 'conditioned stimulus'; e g. the 'conditioned stimulus was inhibited, has undergone extinction', etc. Obviously these are abbreviations for the phrases: 'the *conditioned reflex* to this or that stimulus was inhibited', etc.

impinging upon the same analyser* as the inhibitory stimulus is, *ceteris paribus*, stronger and more prolonged than the inhibition of stimuli impinging upon other analysers.

By way of illustration and better elucidation of the above principles we briefly adduce the results of the experiments carried out by Ivanov-Smolensky and Bieliakov †

Ivanov-Smolensky's experiments. Alimentary conditioned reflexes were established in a dog to the tones: 123 vibrations a second (H), 132 vibrations a second (C), 1036 vibrations (C) and 1161 vibrations (D), and also to a hissing sound and to beats of a metronome. Then, from time to time, one of these stimuli was subjected to acute extinction (by applying it without reinforcement a number of times in succession) and the effect was investigated of the extinguished conditioned stimulus upon the active conditioned stimuli applied after it. The results of these experiments are shown in a diagram compiled on the basis of the author's statistical data (Fig 1). As can be seen from this diagram, the inhibitory after-effect upon conditioned stimuli applied at different intervals after the inhibitor increases gradually during 3–5 minutes, then slowly subsides; the conditioned reflex to the tone nearest to that of the extinguished tone is inhibited most strongly and for the longest time, there is much weaker inhibition to the distant tone, and still weaker inhibition to the other auditory stimulus.

Bieliakov's experiments. An alimentary conditioned reflex was established to a musical tone (auditory stimulus) and to a noiselessly rotating object (visual stimulus). Differentiation was established to half a tone lower. In various experiments the inhibited tone was applied twice or four times, and then after a minute's interval the active tone or the rotating object was tested. After a double application of the differentiated tone the

* The term 'analyser' was introduced by Pavlov to denote the part of the nervous system dealing with a definite kind of stimuli Thus we have the visual analyser, the tactile analyser, the auditory analyser, etc. An analyser possesses a peripheral end (receptors) and a central end (the corresponding receptive area of the cortex).

† Ivanov-Smolensky, A G., *Trudy Lab. Pavlova*, 1924, vol I, p 229 (Pavlov, *Conditioned Reflexes*, pp. 165 et seq) Bieliakov, V. V , Diss , Petersburg, 1911 (Pavlov, *Conditioned Reflexes*, pp 167 et seq).

effect of the conditioned active tone was diminished by 60 % in comparison with the norm, but there was no change in the effect of the rotating object. After a fourfold repetition of the differentiated tone the conditioned reflex to the active tone diminished almost to zero, whereas the conditioned reflex to the rotating object was diminished only by half.

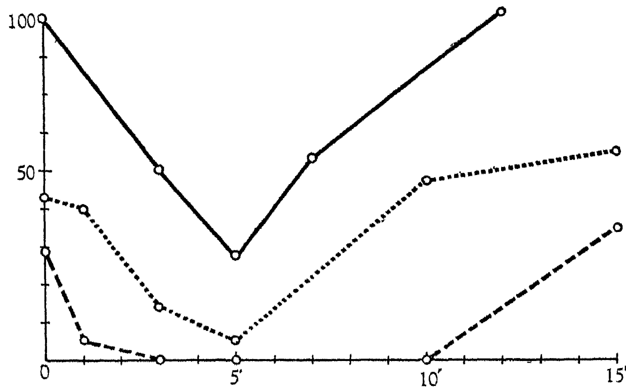


FIG. 1 The dependence of the intensity of inhibitory after-effect upon the time elapsed between the application of the negative and the positive conditioned stimulus and upon the similarity of these stimuli (according to Ivanov-Smolensky's experiments) Abscissa: time (in minutes) elapsed since the last application of the conditioned stimulus submitted to experimental extinction (musical tone of 123 vibrations) Ordinate: the size of the conditioned response to stimuli subjected to the inhibitory after-effect, in percentages of their normal response — — — — musical tone of 132 vibrations (the nearest to inhibitory stimulus) - - - - - - musical tone of 1161 vibrations (more remote from inhibitory stimulus). —— hissing sound.

If we accept the position (as was adopted by Pavlov) that the cortical end of the given analyser constitutes a projection of its peripheral end, in other words that there is a strict point-to-point correspondence between the receptive surface and its cortical representation, then the simplest interpretation of the results above described is as follows: the inhibitory stimulus initiates in its cortical *point d'impact* a state of inhibition which *irradiates* or spreads over the cortex during some seconds, affecting the nearest cortical cells to the greatest extent, the more distant cells of the same analyser to a lesser extent, the

still more distant cells belonging to other analysers to an even lesser extent. Then during some minutes it is *concentrated*, and flows back to its starting-point, successively releasing first the most distant and then the increasingly closer areas of the cerebral cortex. That is the interpretation Pavlov himself gave to the facts we have just described.

Let us consider more closely what is asserted and predetermined by this interpretation. First and foremost, it unequivocally localizes the process of internal inhibition. So far experimental data have not allowed of any precise determination of the particular sector of the conditioned reflex arc in which this process arises; indeed, in the earlier works of the Pavlov school we find continual vacillation as to this point. But if we accept the foregoing interpretation, we have to accept that the seat of the process is the cortical centre of the inhibited conditioned stimulus. In other words, we accept the thesis that under the action of the given conditioned stimulus a state of excitation or of inhibition arises in the cortical cells corresponding to this stimulus, depending on whether it is reinforced and gives a positive reaction, or is not reinforced and so calls forth an inhibitory reaction. From this it follows that in the case of the conditioned stimulus not being reinforced, the given cortical point acquires, either temporarily or permanently, what Pavlov calls 'inhibitory (or negative) excitability', i.e. the property of reacting to stimuli not with a process of excitation but with one of inhibition.

Secondly, we must accept that the state of inhibition arising in a given point of the cortex, because of its negative excitability, does not remain there, but temporarily spreads out into other points of the cerebral cortex, which normally are endowed with 'excitatory (or positive) excitability'.

When it was discovered that inhibition has the property of irradiating over the cortex, it was supposed that this is also true of excitation. This supposition was confirmed by means of two separate categories of facts. One of these categories was obtained in experiments analogous to those of Krasnogorsky.*

* Pietrova, M. K., Diss., Petersburg, 1914. Podkopayev, N. A., *Trudy Lab. Pavlova*, 1941, vol. x, p. 160 (Pavlov, *Conditioned Reflexes*, pp. 177 et seq.).

They consisted in elaborating a positive conditioned reflex to the tactile stimulation of one place on a dog's skin, while other places were differentiated. When differentiation of all these places was well established the experiments proceeded as follows Immediately after the active conditioned stimulus one or another of the inhibitory conditioned stimuli was applied. In these conditions the inhibitory stimuli proved to be dis-inhibited, and the conditioned effect which they evoked was the stronger the closer the stimulated point of the skin lay to the excitatory point. The conclusion this leads to is that the excitation of the given point of the cerebral cortex irradiates and involves other points endowed normally with inhibitory excitability, in consequence of which they become temporarily positive to a greater or lesser extent.

The second category of facts confirming the irradiation of excitation is derived from the phenomenon of generalization of conditioned reflexes. In fact, experimental data show that a conditioned reflex to a tactile stimulation of one place on the skin is generalized to other places, the effect of these secondary conditioned stimuli being the less, the farther the stimulated place is situated from the primary stimulus. If it is assumed that the sensory area of the cerebral cortex constitutes a projection of the surface of the skin, then the phenomenon of generalization can be explained by accepting that the excitation of the given cortical point irradiates into neighbouring points, and these points, being excited simultaneously with the centre of the unconditioned stimulus, link up with it on the basis of the general law of the formation of conditioned reflexes. In the case of differentiation we create in the given analyser not only a focus of excitation, but also one of inhibition; inhibition irradiates over the cortex in the same way as excitation, and thus these processes mutually restrict each other. As the result, after the differentiation of the two stimuli is achieved, the generalization of the conditioned stimulus remains restricted in the direction of the inhibitory stimulus, since the inhibitory centre resists its wider irradiation.

3

We have mentioned that the irradiation of inhibition does not appear immediately after the application of the inhibitory stimulus, but develops gradually, achieving its maximum in the course of some tens of seconds. The question arises, what goes on in the cerebral cortex before the beginning of irradiation? A clear answer to this question was given for the first time by the experiments carried out by Fursikov* several years after the discovery of irradiation He pointed out that the active conditioned stimulus, applied immediately after the inhibitory stimulus, often evoked an effect *greater than usual*, and sometimes almost double by comparison with the normal effect. This fact indicates that, before it has begun to irradiate, the process of inhibition arising in a given point of the cerebral cortex evokes the opposite process, i.e. an increase of excitability of the nerve cells in other points of the cortex. Pavlov called this phenomenon *positive induction*.

Experimental research has been devoted as much to positive induction as to the irradiation of inhibition, and has shown that this phenomenon is far more capricious than inhibitory after-effect, and is less regularly observable. Certain conditions of its manifestation have been investigated fairly precisely. For instance, it has been shown that positive induction arises more easily after differential inhibition than after other varieties of internal inhibition, that it arises with the greater strength and frequency the fresher is the inhibitor (in other words, the less established it is) and the finer the differentiation, and that finally, if the inhibitory reflex is disinhibited by some extraneous stimulus, then positive induction is replaced by inhibition.

By way of illustration we give the following data from Kalmykov's experiments.† From a conditioned stimulus, consisting in a metronome beating at a frequency of 100 a minute, he differentiated a beat at a frequency of 160. When the differ-

* Fursikov, D. S , *Arch Nauk biol* 1923, vol. xxiii, nos. 1–3 (Pavlov, *Conditioned Reflexes*, p 189).

† Kalmykov, M. P , *Trudy Lab. Pavlova*, 1926, vol. i, no. 2 (Pavlov, *Conditioned Reflexes*, p. 191).

entiation was established, he applied from time to time the active stimulus immediately after the inhibitory stimulus, and found that in this situation its effect was regularly greater (by over 50 %). Then a very fine differentiation was established in the same dog, by applying the beat of a metronome at a frequency of 112 without reinforcement. When this differentiation was achieved the new inhibitor evoked a still stronger positive induction (an increase of the conditioned reflex by over 70 %), but the old inhibitory stimulus (160 beats of the metronome) failed to cause any positive induction.

The positive induction appears not only *after* the inhibitory stimulus, but also, to a still greater degree, *during* its application.* This fact shows that during the action of the internal inhibitor the process of inhibition is rather concentrated in the cortical point corresponding to the inhibitory conditioned stimulus, while the irradiation of this process begins only after the stimulus is withdrawn.

In connexion with the facts described in the previous and the present section it is worth while drawing attention to the following state of affairs. If, repeating a conditioned stimulus many times without reinforcement at 2 minute intervals, its effect is reduced to zero, and then at the same interval another conditioned stimulus is tried, owing to the irradiation of the inhibition its effect will be considerably diminished. But if the conditioned stimulus is given *simultaneously* with the extinguished stimulus (or immediately after it) the conditioned reaction may not only be undiminished, but, on the contrary, sometimes shows a certain increase. It follows from this that

* As this fact is not well known, we cite data illustrating it, drawn from Podkopayev's paper (*Jubilee Volume* in honour of Pavlov, 1924, p. 297). The author compared the effect of a conditioned stimulus (a tone) applied *during* the action of a differential inhibitor (beats of a metronome) with the effect of the same stimulus applied immediately *after* the inhibitor. In the case of the tone being presented during the inhibitor, out of nine tests the results were: an increase in the effect of the conditioned stimulus (by 20–36 %) in four tests, the usual effect in four tests, and a slightly diminished effect (by 10 %) in one test. When the tone was given immediately after the inhibitor, out of six tests there was a reduction of the conditioned reaction in four cases (from 30 to 40 %), in one case the reaction remained normal, and in one case there was an increase by 26 %.

whereas after the cessation of the inhibitory stimulus the inhibition irradiates widely over the cortex, on the successive application of this stimulus it is immediately reconcentrated at its starting-point, so releasing the rest of the cerebral cortex from its influence.

Establishment of the fact that a concentrated state of inhibition causes an increase of excitability in the surrounding areas of the cerebral cortex led to the conclusion that, on the other hand, a concentrated state of excitation should produce an inhibition around itself. This principle is called *negative induction*, and many facts of the most varied nature were drawn upon for its confirmation. Here we mention the most important of these.

First, negative induction provided an explanation of all the facts which previously had been called external inhibition. And, indeed, if it is known that an extraneous stimulus evoking an orientation reaction, for instance, inhibits a simultaneously acting conditioned reflex, there is no difficulty in explaining this fact by reference to negative induction. It is assumed that the extraneous stimulus brings about a concentrated excitation in the corresponding point of the cerebral cortex, as the result of which other points of the cortex, including the point corresponding to the given conditioned stimulus, are inhibited.

In the same way excitation of an alimentary centre (evoked by a conditioned or unconditioned stimulus) produces negative induction, as the result of which all the animal's other activities during the act of eating (or waiting for food) are inhibited. This explains, among other things, the fact that, if a stimulus to be conditioned is regularly applied during the action of the unconditioned stimulus, instead of before it, the conditioned reflex, practically speaking, cannot be established,* and, as later research showed, an inhibitory reflex arises in its place.† According to Pavlov this is due to the fact that when the indifferent stimulus reaches the cortical centres during the period

* Krestovnikov, A. N., *Trudy Obshchestva Russkikh Vrachei*, 1913, vol. LXXX (Pavlov, *Conditioned Reflexes*, pp. 27 et seq.)

† Kreps, E. M., *Trudy Lab Pavlova*, 1933, vol. v, p. 5; Pavlova, V. J. ibid. p 21; Vinogradov, N. V, ibid p 33; Petrova, M K, ibid. p. 49 (Pavlov, *Conditioned Reflexes*, pp 391 et seq).

when they are subjected to negative induction it is unable to evoke there the excitatory process necessary for the elaboration of a conditioned connexion.

But in Pavlov's theory negative induction is used to explain not only phenomena of external inhibition, but also other facts in which the excitation in any way strengthens or incites the state of inhibition. For instance, Krzyshkovsky's and Strogonov's investigations* showed that in the course of transforming an inhibitory conditioned reflex to the differentiated stimulus into an active conditioned reflex, the following interesting phenomenon arises. If the inhibitory stimulus is applied successively and reinforced by the unconditioned stimulus, the transformation of this stimulus into a positive stimulus occurs very rapidly. But if this stimulus is applied in alternation with the positive stimulus from which it has been differentiated, the formation of an active conditioned reflex to the inhibitory stimulus proceeds as a rule very slowly and laboriously, and sometimes appears to be almost hopeless. It appears as if the intervention of positive conditioned stimuli between the inhibitory stimuli strengthened the process of inhibition evoked by these stimuli, and hindered the replacing of this process by one of excitation. Pavlov ascribed this fact to negative induction.

In general it has to be pointed out that, as numerous experiments have shown, after a differentiation between two similar stimuli is established, these stimuli become a pair, linked together by inductive relations. For instance, a conditioned response to the positive stimulus usually becomes higher than it was before differentiation, which is ascribed to the constant influence of the positive induction surrounding the inhibitory point; similarly, the application of the positive conditioned stimulus before its inhibitory opposite is favourable to the strengthening of the inhibitory reflex, this being regarded as the result of negative induction, and so on. Particularly clear inductive relations between the excitatory and the inhibitory points were found by Kupalov in his fine experiments concerning the so-called 'cortical mosaic'.† In these experiments positive

* Quoted from Pavlov, *Conditioned Reflexes*, pp. 196 et seq.

† Kupalov, P. S., *Trudy Lab. Pavlova*, 1929, vol. III, pp. 3, 27, 39.

and negative conditioned reflexes were alternately established to the stimulation of several places on a dog's skin The author was able to demonstrate that the processes of excitation and inhibition were in this situation strictly concentrated in the cortical 'points' proper to them and exerted inductive influence on neighbouring points.

Pavlov also considered that relations of mutual induction exist between larger divisions of the nervous system. According to him the cerebral cortex constantly exhibits inhibitory influence upon the subcortical ganglia (negative induction), while the inhibition of the cortex causes excitation of the subcortical ganglia as the effect of positive induction. Similar relations would also exist between the various parts of the cerebral cortex.

4

As can be seen from the foregoing description, in certain cases the states of cortical excitation and inhibition both remain concentrated, causing the phenomenon of induction, whereas in other cases they irradiate to a lesser or greater extent over the cortex. Here we are confronted with the important problem: On what factors does the behaviour of both processes depend? Pavlov was greatly concerned with this problem, and in the last few years of his life he formulated general laws which defined this question. For this purpose he made use chiefly of facts concerning the influence of extraneous stimuli upon active and inhibitory conditioned reflexes.

Now weak extraneous stimuli (like weak light, weak sounds, etc.), unlike strong ones, very often do not cause a diminution of the conditioned reflexes, but slightly increase them,* and they also bring about disinhibition of the inhibitory reflexes This leads to the conclusion that a weak excitation, contrary to a strong one, is not concentrated in the spot where it originates, but irradiates over the cortex, summating with excitations originating in other points, or temporarily 'washing out' the state of inhibition from the inhibitory points. As for stronger excitations evoked by such stimuli as sounds of average strength,

* **Bylina, A. S., Diss., Petersburg, 1910.**

strong light, strong smells, rhythmic touching of the skin, etc., as we have already said, these are concentrated, and consequently they exert an inhibiting effect on the simultaneously acting conditioned reflexes (external inhibition) However, the traces left after the action of such strong stimuli have the same properties as weak stimuli possess *during* their action, i.e. they disinhibit the inhibitory reflexes and increase the active reflexes. From which we can infer that the strong stimulus *after* its cessation behaves in the same way as does a weak stimulus during its action, i.e. it causes irradiation of excitation. The fact that, as was observed long ago (*vide* the works of Pimenov and Grossman),* the trace conditioned reflexes (i.e. reflexes established not to an actual stimulus but to its traces) are generalized much more broadly than ordinary conditioned reflexes, is taken to confirm this view. For in this case, after the cessation of the conditioned stimulus the excitation evoked by it irradiates widely over the cortex, which enables distant cortical points to link up with the centre of the conditioned reflex

Whereas during the action of the strong stimulus excitation is concentrated, very strong excitation behaves like a weak one, i.e. it irradiates at once. Pavlov based this assertion on the following fact. When, during an experiment on alimentary conditioned reflexes a dog was brought to a highly aggressive state towards an outsider present in the room, the conditioned reflexes grew considerably, this being explained by the irradiation of the excitation from the 'centre of aggression' to other centres, *inter alia* to the alimentary centre. But when the animal quietened down a little and only fixed its gaze on the stranger, the conditioned reflexes, on the contrary, grew less than the normal, since the somewhat weakened excitation in the 'centre of aggression' became concentrated and began to cause a negative induction in other centres (Bezbokaya's experiments).†

A similar state of dependence between the strength of a process and its diffusion is to be observed in the process of inhibition.

* Pimenov, P. P., Diss., Petersburg, 1907. Grossman, F. S., Diss., Petersburg, 1910 (Pavlov, *Conditioned Reflexes*, p. 113).

† Bezbokaya, M., Diss., Petersburg, 1913 (Pavlov, *Conditioned Reflexes*, p. 182).

When the inhibition is weak, i.e. incomplete (not fully formed), it does not usually give a positive induction, and so the inhibitory stimulus exerts an inhibiting effect upon the conditioned stimuli even during its actual duration. This shows that the process of inhibition irradiates at once, without previous concentration. In the case of a stronger inhibition, when it is wholly formed and complete, during the action of the stimulus and immediately afterward a positive induction is observed, i.e. there is a concentration of inhibition, which later yields place to irradiation. Finally, a very strong inhibition, which is obtained through a marked prolongation of action of a differential inhibitor, or through the manifold repetition of the inhibitory stimulus, again gives almost no positive induction, but only a considerable weakening of the active conditioned reflexes, i.e. a strong irradiation.

All the facts above adduced led Pavlov to formulate the following general statement: 'with a slight tension of either the excitatory or inhibitory process, under the action of corresponding stimulation, irradiation carries the processes from the original point; with a moderate tension it is concentrated in the original point, and with marked tension, there is again irradiation'.*

According to Pavlov sleep is a demonstrative example of the irradiation of inhibition. The phenomenon of sleep was met with quite early on in experiments on conditioned reflexes, and it was established that it frequently occurs in strict association with internal inhibition. Thus, the deep extinction of a conditioned reflex, or even of an orientation reflex, or the manifold successive repetition of a differential inhibitor, often leads to the animal falling asleep. Similarly, with a greatly delayed conditioned reflex, when the reinforcement follows only after the conditioned stimulus has continued for 3 minutes, the dog frequently falls asleep in the inhibitory phase, i.e. in the first 2 minutes, and wakes up in the third minute, not long before the food is given. In all these cases we have inhibition not concentrated, but strongly irradiating. But, on the other hand, the application of a well-established differential inhibitor alter-

* Pavlov, *Lectures*, vol. II, p. 87.

nately with positive conditioned stimuli favouring a concentration of the inhibition and positive induction, never causes sleep. On the basis of these data Pavlov came to the conclusion that 'internal inhibition and sleep are one and the same process'; in other words, sleep comes as the result of a strongly diffused state of inhibition, which involves not only the cerebral cortex, but the subcortical ganglia.

During the first phase of the act of falling asleep a certain motor excitation and some enhancement of subcortical reflexes are often observed. Pavlov explains this as due to the inductive relations existing between the cortex and subcortical ganglia, which cause the subcortical centres, which have been partially inhibited during the active state of the cerebral cortex (negative induction), to be excited during the inhibition of the cortex (positive induction). Only when the sleep is deeper does the irradiation of the inhibition spread to the subcortical ganglia. According to Pavlov hypnosis is a state of partial sleep, when little islands of excitation emerge against the background of the inhibited cortex.

Confirmation of the above conception is found in the facts that indicate that a deepening of inhibition often leads to sleep, while, on the other hand, a weakening of the inhibition, or its concentration, eliminates sleep. For instance, experiments are conducted on conditioned reflexes in which the reinforcement is postponed for 30 seconds. It is observed that during the experiment the animal is sleepy, and more and more frequently falls off to sleep completely for longer and longer periods. It is only necessary to reduce the isolated periods of the conditioned stimulus to 5 or 10 seconds (whereby the inhibition of delay is eliminated) or to introduce a differential inhibition and elaborate it satisfactorily (whereby we intensify the concentration of the inhibition) for the drowsiness and the sleep to vanish. Many similar examples could be adduced.

The results of experiments carried out by Galkin and Speransky, who established that dogs deprived of three receptors (visual, auditory and olfactory) spend almost all their time in sleep, led Pavlov to the assumption that besides active sleep, as above described, there is also 'passive' sleep, caused by the

absence of stimuli to the cerebral cortex.* But later he came to the conclusion that 'passive' sleep possesses the same mechanism as 'active' sleep, and that the sleep of animals deprived of receptors is also caused by inhibition, which is the result of monotonous stimuli originating from the tactile receptors. The strong irradiation of this inhibition is due to the absence of excitation in other areas of the cortex, in consequence of which the process of inhibition meets with no obstacles whatever in the course of its dispersal.†

5

As we have seen from the preceding sections, the second stage of the work on the physiology of the higher nervous activity was chiefly devoted to research into the interactions between various reflexes, both conditioned and unconditioned, both active and inhibitory. These investigations led to the establishment of laws for the 'dynamics' of the cortical processes, namely, of the law of irradiation and concentration of the processes of excitation and inhibition, and that of mutual induction. During the third decade of this century this stage began imperceptibly to pass into the next, which has dominated all the work on conditioned reflexes ever since In this last period the chief role is given over to the investigation of the functional properties of various 'points' of the cerebral cortex under the influence of various factors.

It has long been known that if, on the basis of a defined unconditioned stimulus (for instance a regular portion of food given), conditioned reflexes are elaborated to the various stimuli, each of these reflexes develops to a certain magnitude, where, given unvarying experimental conditions, it remains constant. However, this magnitude is different for different stimuli. The stronger the conditioned stimulus, the greater the conditioned reflex it evokes. For instance, a stronger tone acting as a conditioned stimulus gives a greater response than a weaker tone, a higher frequency of the metronome beat gives

* E g Pavlov, *Lectures*, vol. II, p. 89.

† Pavlov, 'The problem of sleep', in the first volume of his *Collected Works*, p. 409, Moscow, 1940

a greater response than a lower frequency. Furthermore, it has been observed that in general intermittent agencies are stronger conditioned stimuli than continuous agencies, and the acoustic stimuli used in the laboratory produce in general a greater conditioned effect than tactile or visual stimuli. All this class of phenomena has been pretty thoroughly worked over experimentally, and these investigations led to Pavlov's formulation of the 'law of the relationship between the magnitude of the effect and the strength of stimulation', which lays down that the more strongly a cortical 'point' is stimulated, the stronger is the conditioned effect. Thus the magnitude of the conditioned reflex becomes an indicator of the strength of excitation of the given cortical point.

However, further investigations revealed that the strength of excitation of the cortical cells depends not only on the strength of the stimulus acting on them, but also on two fundamental properties of these cells, namely, on their *excitability* and their *capability*. In every case both these properties determine the magnitude of the conditioned reflex to the given stimulus.

The greater the excitability of the cortical cells, the stronger, *ceteris paribus*, is the conditioned reflex to the given stimulus, provided there is no obstacle in the too poor capability of these cells (see below) According to the Pavlov theory small injections of caffein, moderate hunger, and certain other factors, cause an increase of reflexes through an increase in the excitability of the cortical cells. On the contrary, the satiation of the animal causes a diminution in the excitability of the cells and a reduction in the conditioned reflexes. Old age, the removal of the thyroid gland, etc., have a similar but chronic effect.

The concept of the capability of the cortical cells arose in connexion with the following type of experiment. If conditioned reflexes are elaborated to various stimuli from the weakest to the strongest, it is found that whereas strong stimuli evoke the maximal conditioned response, very strong stimuli, so-called supra-maximal, evoke an effect more or less diminished. According to Pavlov the explanation of this is that the cortical cells possess

a 'top capability',* in other words, they are capable of reacting by excitation to the stimuli that reach them only up to definite limits. If the stimulus exceeds the top capability of the corresponding cortical cell, this cell reacts to such a stimulus with inhibition, the so-called *top* or *protecting inhibition.* According to Pavlov and his followers, the biological role of this inhibition is by its intervention to prevent damage to the cortical cells which might result from too strong an excitation, i.e. to protect the cells from the strain of too great a 'loss of energy'.†

The capability of the cortical cells depends on many factors, and may change either acutely or chronically. First of all it depends on the type of animal. The cortical elements in animals with a so-called weak nervous system are of poor capability, i.e. in their case any strong excitation easily becomes supramaximal and leads to top inhibition. Further, such factors as neuroses, old age, and, among transient factors, drowsiness also weaken the cortical cells, i e. they lower their top capability.

It is easy enough to realize what consequences follow from a lowering of the top capability of cortical cells. A strong conditioned stimulus which had previously been evoking a maximal conditioned reaction, as the result of a reduction of the cortical cells' capability, becomes a supra-maximal stimulus, and as the result of the top inhibition its effect is diminished, becoming equal to the effect of weaker stimuli. This is what is called the *phase of equalization,* when both strong and weak conditioned stimuli evoke an identical conditioned reaction. With a still greater lowering of the top capability the effect of strong

* I prefer the word 'top' to the word 'limit' used by Gannt in his translation, as more precisely corresponding to the Russian word 'priediel'. 'Zapriedielnoye Tormozhenie' I translate 'top inhibition' J K.

† The attentive reader will certainly see a contradiction between the statement that very strong stimuli evoke top inhibition, and the earlier statement that they evoke strong excitation irradiating over the cortex This contradiction (which has not been explained either by Pavlov or by his followers) is in our view to be explained within the framework of Pavlov's theory as being due to various nerve cells possessing various tops of capability. Cortical cells, which have no great capability, react to strong stimuli with top inhibition, whereas subcortical cells, which have a far greater capability, react to such stimuli with an adequately strong excitation, which because of its strength overflows into other centres. It seems to us that this explanation is completely in accordance with the spirit of Pavlov's theory

stimuli may become less than that of weak stimuli, which state is called the *paradoxical phase*. With a very considerable reduction of capability the so-called *ultra-paradoxical phase* is sometimes observed, when positive conditioned stimuli are completely inhibited, whereas the inhibitory stimulus evokes a positive conditioned reaction. The explanation of this phase is that the inhibited excitatory point produces excitation in the inhibitory point, with which it is linked by mutual induction.

The phase of equalization and the paradoxical phase can be obtained not only by lowering the top capability of the cortical cells, but also by an increase in their excitability. In these conditions the maximal stimulus may evoke too strong an excitation, causing a top inhibition, in consequence of which its effect becomes equal to the effect of weaker stimuli, or even less. This explains why large doses of caffein produce an increase of conditioned reflexes to weak stimuli, whereas strong stimuli, on the contrary, are weakened, which results in an equalization or paradoxical phase.* Similar relations between conditioned reflexes are sometimes met with in cases of hunger.†

Animals with a so-called weak nervous system, i.e. with cortical cells of a poor capability, always exhibit disturbance of the law of strength of stimuli, in the form of the phases above mentioned. In normal animals such disturbance is manifested in states of drowsiness (hence the term 'hypnotic phases'). As we have said, there is a chronic diminution in the capability of cortical cells in the case of experimental neuroses, which have been very thoroughly investigated by the Pavlovian school.

Experimental neuroses are pathological states of the cortical activity, evoked in a purely functional manner, i.e only by certain combinations of stimuli which are detrimental to the animal's nervous equilibrium. Among the factors that can evoke experimental neuroses are: (1) collision of the processes of excitation and inhibition, produced by the application of an active and an inhibitory conditioned stimulus concurrently or

* Kleshchov, S V, *Trudy Lab. Pavlova*, 1938, vol. VIII, p. 182 Zewald, L. O., ibid p. 369.

† Rikman, V. V., unpublished experiments. Vide Pavlov, *Lectures*, vol. II, pp. 54 et seq.

in immediate succession; (2) overstrain of the process of inhibition, when, for instance, the duration of the differential inhibitor is protracted from its usual 15–30 seconds to several minutes, (3) overstrain of the process of excitation, evoked by supra-maximal stimuli; and finally (4) any 'task' too difficult for the animal's nervous system, e.g. a very subtle differentiation, the application of the same stimulus sometimes with and sometimes without reinforcement, etc. The last factor may be regarded as a collision between excitation and inhibition

We may cite the neurosis obtained in Razienkov's experiments as a classic example of such a state with all its typical symptoms.* This experimenter produced a collision of the process of excitation and inhibition in a dog, by applying a positive conditioned stimulus (rhythmic stimulation of a given point on the skin at a frequency of 24 touches per minute) immediately after a negative stimulus (rhythmic stimulation of the same spot on the skin at a frequency of 12 to the minute). From the following day onward over the course of a few weeks the conditioned reflex activity of the animal suffered considerable disturbance. During the first 9 days the conditioned reflexes were down to zero; then they began to appear, but the relationship between the strength of the stimuli and the conditioned effect was reversed, i.e there occurred a paradoxical phase which lasted 2 weeks, then came an equalization phase, and only after it had passed did the dog gradually return to normal.

In bad cases of neurosis pathological phenomena are met with also in the sphere of behaviour, e g. despite its hunger the dog does not take the food offered, displays negativism and ambivalence, cataleptic states, etc. As these phenomena belong to the pathophysiology of the cerebral cortex, they lie beyond the scope of the questions with which we are concerned in this book

6

In this section we desire to give an exposition of Pavlov's views on the nature of the process of cortical inhibition and the state of 'inhibitory excitability' of the cortical cells. This problem

* Razienkov, I. P , *Trudy Lab Pavlova*, 1924, vol. I, no. 1 (Pavlov, *Conditioned Reflexes*, pp. 270 et seq).

greatly occupied him; he called it the 'damnable problem' of physiology of the higher nervous activity, and put forward a number of hypotheses regarding it, but finally could not decide in favour of any, since they all seemed to him imperfect in some respect and none of them could cover all the facts they were intended to explain. Here we give the most important of these hypotheses together with the defects which made them unsatisfactory to Pavlov.

Quite early on the view was accepted by the Pavlov school that inhibition is the opposite state to excitation, that it is a kind of 'negative excitation'. Therefore there was a continual search for a symmetry between the two processes, and a tendency to find in every property of one process the converse of the corresponding property of the other. Thus the irradiation of inhibition stimulated investigations into the irradiation of excitation, and positive induction was the source of the conception of negative induction In this connexion the discovery of top inhibition was a serious breach in the idea of the symmetry of the two processes, for no corresponding phenomenon for excitation was ever found.

As excitation is a kind of discharge of energy of the nervous cells, in other words is associated with the catabolic process, Pavlov associated inhibition with the opposite process, of reconstruction or assimilation. And so he very often expressed the opinion that inhibition is a rest for the cortical cells, of which the most demonstrative expression he considered to be the recuperative action of sleep. It is this that led Pavlov to give his approval to the sleep treatment of nervous diseases. But, on the other hand, he realized the great difficulties that are inherent in this conception. For the accumulated evidence concerning internal inhibition indicates that it is an 'active' process, that it has to be established often long and laboriously, that its maintenance demands specific 'cerebral work' on the part of the animal, that too frequent repetition of inhibitory stimuli lead to their disinhibition, and so on. So how in such cases can one speak of a rest for the cells of the cerebral cortex, when experimental data show something rather diametrically opposite?

There is a certain connexion between the assimilation theory of inhibition and the conception, according to which inhibition, or more strictly 'inhibitory excitability', of a given cortical point is associated with its functional exhaustion. One of the main facts on which this theory is based is that the frequent repetition of one and the same conditioned stimulus leads, even though it is reinforced each time, to a gradual diminution of its effect, which may even fall to zero. This occurs either rapidly, in one experiment, or chronically, in a long series of experiments * This phenomenon is called 'extinction with reinforcement'. Pavlov explained it as follows. Every cortical cell which is excited many times over becomes exhausted, and so passes into a state of inhibition. So the problem is not why does a state of inhibition arise in the cortical cells, but why does the non-reinforcement of a conditioned stimulus lead to this state incomparably more rapidly than does the repetition of this stimulus with reinforcement. The answer is based on the above-formulated theory of the anabolic role of inhibition The application of an unconditioned stimulus (e g. offering food to a dog) evokes concentrated excitation surrounded by strong inductive inhibition in other parts of the cerebral cortex, and as the result a conditioned stimulus acting simultaneously evokes inhibition in its *point d'impact* (cf. p. 19). This inhibition is the very factor that enables these cells to recover.

In connexion with this conception experiments were made which tended to show that the more the conditioned stimulus overlaps the unconditioned stimulus, the stronger is the conditioned reflex. This conception also explains why a conditioned reflex weakened by too prolonged training shows marked recovery if the isolated period of conditioned stimulus is shortened.

However, in this conception also Pavlov saw great difficulties. He formulated them as follows: 'It is obvious that only certain cases of the development or disappearance of inhibition can be brought into relation with a supposed functional exhaustion and recovery of the cortical elements, and we cannot interpret in this fashion the cases of permanent and unvarying inhibitions

* On this question see Pavlov, *Conditioned Reflexes*, lecture XIV.

in which the activity of the cortex is so rich—for example, all cases where an established inhibitory conditioned stimulus evokes an inhibition of the cortical elements directly and without a preceding phase of excitation, as, for instance, in the case of differentiation and conditioned inhibition The problem presented by inhibitions of the latter type becomes still more complicated when we remember that those points of the cortex which become the centres of such direct inhibitions are never transformed into centres for excitation, even though the experiments are interrupted for weeks and months.'*

In the last years of his life, Pavlov put forward a new conception of inhibition which it seems was never formulated by him in writing.† According to this conception the state of inhibition in the cortical cells consists simply in a reduction of their capability. Thus, if some cortical 'point' is constantly inhibitory (i e. is a *point d'impact* for an inhibitory stimulus), this is because its capability is almost down to zero, and consequently it responds to every stimulation only with the process of inhibition. The irradiation of inhibition is nothing but acute and transient lowering of the top capability of all the cells in the cortex because of the action of an inhibitory stimulus. If the reduction of capability is not very significant, there is a diminution in the conditioned reflexes, which results in corresponding 'hypnotic phases'. If it is considerable, the inhibition is complete. An acute reduction of capability arises also in cortical cells through negative induction, evoked by a concentrated focus of excitation. And so all kinds of inhibition would be reduced to top inhibition.

We do not know why Pavlov did not develop this conception to any extent. Possibly it was because he did not wish to break altogether with the idea of symmetry in the processes of excitation and inhibition, which would have to be rejected *in toto* if this conception were accepted. Possibly he also saw other insuperable difficulties in the way of its acceptance.

* Pavlov, *Conditioned Reflexes*, p. 249

† We find a vague reference to it in the lecture in Rome, 1932.

7

In concluding this sketch of Pavlov's theory (which of course we do not claim to be complete) we have to state the following·

This theory is the fruit of the great Russian physiologist's many years of 'incessant meditation' on a vast amount of experimental work performed by a large group of his collaborators, over a period of more than 30 years. Those acquainted with the literature covering the physiology of higher nervous activity, and even more those who made acquaintance in Pavlov's own laboratories with the vast accumulation of *unwritten* knowledge in this field, know what a mighty instrument his theory was in his hands, how keen and cogent was his analysis on its basis not only of experiments on dogs but of psycho and neuropathic phenomena in human beings, and how greatly the theory stimulated further experimental work. I think, however, that the apparent harmony and beauty of this theory was a little too seductive and, held in its power, we failed to discern many discrepancies between the theory and the facts, we closed our eyes to its seemingly tiny inconsistencies Perhaps the above mentioned properties of the Pavlov theory were responsible for the circumstance that as the years passed it was not only never revised but, on the contrary, more and more crystallized, and finally became a dogma, inhibiting the development of this branch of physiology for many years.

Chapter III

A criticism of the Pavlov theory

1

Before we enter upon a criticism of the Pavlov theory, we wish to make it clear that we have no intention of engaging in a detailed analysis of a variety of Pavlov's pronouncements and picking upon any inaccuracies to be found in them, or upon discrepancies between one pronouncement and another, such as we shall undoubtedly find if we go through all his works over the thirty years of his investigations into conditioned reflexes. Nor have we any intention of captiously questioning various expressions or terms used by him and of proving that they can be replaced or improved upon, nor of making use of any experimental data which have not been adequately verified, nor, finally, of examining and criticizing hypotheses which Pavlov himself regarded as provisional and inadequately justified. We know Pavlov's mental grasp and scope, and how frequently he modified and changed his views in the light of new facts. And because of this there is nothing easier than to submit his work to petty and unfair criticism (such as is sometimes found in the literature of this subject) concerned with combating clumsy formulations or unessential details of the great scientist's conception. Such criticism does not contribute anything of positive value to the field of knowledge we are surveying. Therefore we shall base our criticism only on such experimental data as constitute a permanent scientific acquisition in regard to conditioned reflexes, and shall take into consideration only those of Pavlov's conceptions which he continued to affirm to the end, and which constitute the core of his theory.

Pavlov's theory can be submitted to critical analysis from two viewpoints. First, we can investigate whether and to what extent it is in harmony with the data and conceptions of the modern physiology of the central nervous system. Secondly, we can seek to elucidate whether it contains any intrinsic contra-

dictions, and whether its statements are in harmony with the experimental facts on which they are based.

2

We have no intention of devoting much space to a criticism of the Pavlov theory from the viewpoint of data drawn from contemporary physiology of the central nervous system. For, on the one hand, the previous chapter shows quite clearly the extent to which the whole *style* of this theory is foreign to contemporary physiological thought concerning the nervous system, and so a detailed specification of the discrepancies that exist is quite unnecessary. On the other hand, we must bear in mind that study of the physiology of higher nervous activity is so complex and difficult that, if anyone formulated a system of working hypotheses which completely accorded with the factual evidence and were not internally contradictory, we should be prepared to accept that system, even if it departed considerably from the system of conceptions developed in the physiology of other parts of the nervous system.

The essential difference between the two approaches concerns on the one hand the nature of the processes, and on the other their propagation over the nervous tissue.

We shall first consider the question of the nature of the nervous processes, concentrating all our attention on the process of inhibition, in which the difference between the two approaches is particularly obvious. As we know, according to the evidence of contemporary physiology, inhibition is a *synaptic* process. This means that, quite independently of the theory we adopt for the explanation of this phenomenon, it is considered that it goes on at synapses and concerns the transfer of a nervous excitation from one neuron to the next. If a reflex is inhibited, this is because in one of the intermediary stations of its reflex arc a block of nervous impulses occurs, and in consequence the neurons receiving the impulses are unable to transmit the excitation to their axons.

From the viewpoint of the Pavlov theory the process of inhibition is totally different. According to this theory two

different forms of this process may be distinguished, though there is by no means a strict demarcation between them.

In certain cases the cortical cell possesses *sui generis* 'inhibitory (negative) excitability', i.e. a certain more or less lasting property, thanks to which, stimuli reaching this cell always initiate a process of inhibition This happens when a permanent inhibitory conditioned reflex has been established to a stimulus, e.g. by differentiation, or by chronic extinction. Thus the cerebral cortex contains points endowed with 'inhibitory excitability' and 'excitatory excitability', and it represents, in Pavlov's words, 'an enormous assemblage of positively and negatively excitable points thickly and patchily intermingled'.

In other cases the process of inhibition arises in cortical cells not predestined to this role, namely, it is passed on to them, or flows over into them for some reason or other. This happens with irradiation of inhibition, with negative induction, and also under the influence of supra-maximal stimuli.

In both cases inhibition is a process taking place *in the cortical cells themselves*, and depends, as Pavlov assumes, on their metabolism. Just as, according to him, excitation is connected with catabolic processes of the cell, so the anabolic processes correspond to inhibition. The gulf between the two conceptions we have just presented is so obvious that there is no need to go into the matter further.

There is a similar disparity in regard to the transmission of the nervous processes over the nervous tissue. The evidence of neurophysiology shows that there is one unquestioned form of such transmission, i.e. the transmission of excitation from neuron to neuron by means of nervous impulses. No facts seem to exist at present which would necessitate our going beyond the neuron theory of the functioning of the nervous system and would compel us to accept quite different forms of propagation of the nervous processes. And yet the Pavlov theory is concerned almost exclusively with such different forms. Only, perhaps, the course of the conditioned reflex itself is subordinated to the general scheme of nervous activity. But where irradiation and concentration of excitation and inhibition as well as their mutual induction are concerned, the substratum of these processes is

by no means the differentiated network of neuronic structure but some kind of cell continuum (which, it should be mentioned, is not completely homogeneous) in which the processes flow and ebb or mutually restrict one another but do not travel along any definite paths.

It is true that by special effort and making various corrections to and modifications of the Pavlov theory, it would be possible somehow or other to bring isolated statements of his within the scheme of neuron activity. Thus, the Pavlovian irradiation of excitation could in certain instances be treated as a spreading of this process from neuron to neuron along the nerve paths, and concentration of this process could be regarded as simply its gradual disappearance. The irradiation of inhibition could be explained on the basis of Lorente de Nó's evidence that the cerebral cortex consists of closed chains of neurons in a state of continual activity; inhibition in one chain might involve a depression of activity in the successive chains, and so the 'wave of inhibition' would proceed further and further Negative induction would correspond to the inhibitory influence of higher centres on lower, while positive induction would be analogous to the 'release phenomenon'. But while it might perhaps be possible to reconcile these phenomena (taken separately) with the known mechanisms of nervous activity, their interrelations and interaction are, in the Pavlov theory, something absolutely specific. The processes of excitation or inhibition, which in certain cases are concentrated and surrounded with processes of the opposite 'sign', but in other cases break down this wall of induction and flow more or less broadly, and the dependence of the state of the given process on its strength—all this, in our view. cannot be brought within the framework of the neuron theory.

We repeat that we do not attach much importance to this part of our criticism, because it would be convenient to accept Pavlov's system of conceptions in their entirety (at least as temporary working hypotheses) if they were capable of adequately explaining and systematizing the existing factual data, and did not contain internal contradictions. But in the following sections we shall attempt to show that Pavlov's theory cannot satisfy us in any of these regards.

3

As we have said in the preceding chapter, the fundamental feature of Pavlov's theory, which sets its mark on it and determines all its statements, is the assumption that not only the process of excitation evoked by the application of an active conditioned stimulus, but also the process of inhibition, evoked by the application of an inhibitory conditioned stimulus, is localized in the cortical centre of this stimulus, in the point of the cerebral cortex to which it is 'addressed'. This assumption, which came to be formulated at the end of the first decade of work on conditioned reflexes, took deeper and deeper root with the course of time, and finally became something indubitable and not open to discussion, a dogma on which, as we saw in the previous chapter, all the conceptions of the Pavlov school concerning the activity of the cortical processes are based. This assumption predetermines that all the fundamental processes which occur in the cerebral cortex when positive and inhibitory conditioned reflexes are in action, when they mutually interact, when they are influenced by extraneous stimuli, etc. — all these processes take place at the very beginning of the cortical part of the corresponding reflex arcs, and that one or another conditioned reaction, or the absence of it, is only to be regarded as an indicator which gives notice of the 'sign' and intensity of the process. Thus the reflex arc as a whole disappears completely from sight, and attention is concentrated on unspecified states of excitation and inhibition, irradiating or concentrating, summating or mutually restricting—unspecified because it does not matter in the least which executive neurons they are linked to.

In our view this assumption is the 'original sin' of all Pavlov's theory, for it constitutes the turning point in the development of the physiology of the higher nervous activity, from which research in this field was directed along wrong paths.

The inadequacy of this assumption can be readily seen as soon as we free ourselves of thinking in terms of the Pavlov theory. In fact, the stimulus which we (or the animal) perceive is for us *one and the same stimulus* irrespective of whether it is accompanied by an unconditioned stimulus or not, whether it evokes

a secretion of saliva, or whether it has ceased to evoke such a secretion in consequence of non-reinforcement. Therefore it seems quite natural to assume that one and the same nervous process arises in the cortical centre of the stimulus irrespective of whether it plays the role of positive or negative conditioned stimulus. And yet, accepting the Pavlov theory we are compelled to believe that the same stimulus gives rise to one cortical process (viz. that of excitation) when it is reinforced, and to a quite different process (viz. that of inhibition) when the reinforcing stimulus is omitted.

The same kind of difficulty arises when top inhibition is considered. According to the Pavlov theory, if a stimulus is extraordinarily strong, then besides excitation (or instead of it) protective inhibition arises in the cortical centre, for the purpose of safeguarding the cortical cells from damage caused by too intense an excitation. But, after all, there is not the least doubt that the animal perceives such a stimulus (just as we do) as extraordinarily strong, bears it with difficulty and usually is afraid of it, frequently the dog trembles, runs from the stand, will not take food, etc What right have we to assert that a state of inhibition arises in the centre of the stimulus itself for the sole reason that the conditioned reflex to it is diminished?

Like the basic Pavlovian conceptions, so, too, their direct consequences are often quite unacceptable. For instance, Grigorovich and Yaroslavtseva* studied the question of the character possessed by the intermediary stimulus between the active conditioned and the differentiated stimuli, in order to elucidate what happens on the border between the areas of points charged positively and others charged negatively. Both authors reported that between the two areas there is an indifferent field, i.e. stimuli reaching it evoke neither excitation nor inhibition. Podkopayev came to similar conclusions on the basis of his experiments.† He found that an extraneous stimulus of long duration has neither excitatory nor inhibitory influence on conditioned reflexes, from which he drew the conclusion that this

* Grigorovich, L. S., *Trudy Lab. Pavlova*, 1932, vol. IV, p. 224, Yaroslavtseva, O. P., ibid p. 258.

† Podkopayev, N. A., *Trudy Lab. Pavlova*, 1945, vol. XII/2, p 170

stimulus produces in the cortex neither excitation nor inhibition, only 'an indifferent state'.

How a stimulus reaching the cerebral cortex of a wakeful animal, and undoubtedly perceived by the animal, has no effect whatever in the cortex, or brings about some mysterious 'indifferent state', is for the authors of these experiments to elucidate.

As we write now about the above-mentioned defects they seem to us so obvious that it is rather difficult not to perceive them. And yet even to-day many of Pavlov's disciples and followers, hypnotized by his theory, either do not see, or else do not want to see them. It is difficult to resist the impression that fate has taken a mischievous revenge on Pavlov because, having discovered facts which served as a foundation for his theory, he resolutely turned his back on all attempts at their psychological understanding, was contemptuous of introspection, and deliberately rejected it.*

4

We shall now show that the laws of cortical activity established by Pavlov cannot be maintained because of the internal contradictions they contain.

First and foremost it must be pointed out that at the very bases of the Pavlov theory is an essential conceptual vagueness, which no one has ever tried to remove, and which, it seems to us, cannot be removed. We refer to the distinction between the concepts 'excitation' and 'positive excitability', and between the concepts 'inhibition' and 'negative excitability'. 'Excitation' and 'inhibition' are actual processes occurring in the nerve cells, while 'positive excitability' and 'negative excitability' are certain permanent properties of these cells, responsible for their reacting with excitation or inhibition to the stimuli reaching them. Yet, in the Pavlov theory these concepts are confused, and very often one of them is incorrectly used for another.

* Vide Pavlov, 'The pure physiology of the brain' (*Lectures*, vol. I, p. 245), 'Physiology and psychology in the study of the higher nervous activity of animals' (*Lectures*, vol. I, p. 261) and elsewhere.

We shall give some examples of this confusion.

As we have said in the preceding chapter, after the differentiation of two similar stimuli is established, each of them gives rise to a process of excitation or inhibition concentrated in a small area and mutually restricting each other. But at the moment when the positive conditioned stimulus acts in the points with negative excitability the process of inhibition is inactive, just as at the moment when the inhibitory stimulus acts there is no actual excitation in the points with positive excitability. So it is not clear how one process can be restricted by the opposite process which at that moment is not functioning. On the other hand, there is evidence that inhibitory excitability does not constitute any obstacle to the spread of the process of excitation, just as excitatory excitability does not hinder the irradiation of the process of inhibition.

Similarly, it is known that after the establishment of differentiation the effect of the active conditioned reflex becomes greater, which is explained by the permanent influence of positive induction coming from the inhibitory focus But positive induction can be brought about only by the actual process of inhibition, and not simply by the existence of an inhibitory point. So long as it is not stimulated this point surely cannot exert any influence, whether excitatory or inhibitory, on other points. And yet the increase of the reflex to the conditioned stimulus is permanent, and does not in the least depend on whether a differentiated inhibitory stimulus has or has not been applied previously. Similarly, it is often said that the administration of bromide increases the size of conditioned reflexes in consequence of the concentration of inhibition caused by this drug, and the positive induction this concentration evokes. Inhibition, however, can be concentrated or not only when it actually occurs, whereas the increase of conditioned reflexes after corresponding doses of bromide occurs even when we do not apply any inhibitory stimulus

In all these examples the Pavlov theory unjustifiably makes use of the actual processes of excitation and inhibition to explain facts that concern only positive and negative excitability.

Moreover, it must be pointed out that in many instances it

is not possible at all to define whether we have to do with an actual nervous process, or only with a corresponding 'attitude' of nerve cells, and frequently one or the other of these concepts is used in accordance with convenience. For instance, when considering the irradiation of excitation due to generalization, it must be assumed that in this case the actual process of excitation is spreading, for otherwise the nerve cells situated around the point originally excited could not form conditioned connexions with the centre of the unconditioned stimulus. Similarly, if a wave of excitation (caused by a weak foreign stimulus) summates with the actual excitation of the centre of a conditioned stimulus, then it is understood that the actual process of excitation is spreading over the cortex. But in other cases we find quite another state of affairs. For instance, the disinhibition of inhibitory reflexes by weak extraneous stimuli is understood as meaning that a wave of excitation 'washes away' the inhibitory excitability of the given point, and confers on it a transient positive excitability. Again, in Pietrova's and Podkopayev's experiments (cf. p. 15), excitation irradiating from a point with positive excitability to points with negative excitability temporarily transforms them into points endowed with positive excitability. So here there is no talk of an actual excitation of these points, but only of a change in the sign of their excitability.

An analogous lack of clarity can be found in the analysis of different cases of inhibition. It is known that after the elaboration of a permanent inhibitory reflex to some stimulus (e.g. by differentiation) the corresponding cortical 'point' acquires an 'inhibitory excitability', and in consequence stimuli reaching it give rise to a process of inhibition. Analogously it can be assumed that the same situation is to be found in acute extinction, and that there, too, the centre of the conditioned stimulus temporarily acquires negative excitability, which lasts until the conditioned reflex has recovered. But, on the other hand, it is known that an actual process of inhibition continues in this centre throughout the whole period of extinction, irradiating or concentrating in dependence on the moment, and it is the cessation of this process that marks the recovery of the conditioned reflex. Similarly, it is impossible to decide which of

the two states is imparted to the cortical cells during the irradiation of inhibition or negative induction, since certain facts suggest that there is an actual process of inhibition, while others compel the assumption that there is only a state of negative excitability.

But we shall try to avoid this conceptual vagueness, assuming that there is no strict demarcation between excitation and positive excitability, or inhibition and negative excitability, so that analogous properties *can* be ascribed to both these phenomena, and we shall show that, even on this assumption, the laws of the functioning of the cerebral cortex as adopted in the Pavlov theory cannot be maintained.

5

We shall begin with an analysis of the properties of the process of cortical excitation. We recall that on the basis of the fact of generalization on the one hand, and of the experimental data obtained by Pietrova and Podkopayev on the other (cf. p. 15), a law of irradiation of excitation over the cerebral cortex was formulated. And on the basis of data concerning the influence exerted by extraneous stimuli on conditioned reflexes (cf. p. 21 et seq.) it was concluded that weak excitations and very strong excitations irradiate over the cortex during the action of the stimulus, whereas those of moderate strength are concentrated, and their irradiation begins only after the stimulus has ceased to act. It can be demonstrated that all these inferences, which apparently confirm one and the same law, that of the irradiation of excitation, really contradict one another.

We shall attempt first to compare the experimental data obtained by Pietrova and Podkopayev with the phenomenon of generalization. According to the law of generalization the irradiation of excitation spreads as far as the generalization extends. Thus, if by stimulation of one point on the dog's skin with reinforcement and other points without reinforcement the differentiation in the tactile analyser is established, the inhibitory points in the cortex restrict the irradiation of the excitation and so confine the generalization to the zone in the proximity of the place stimulated with reinforcement. However, as Pietrova's

and Podkopayev's experiments demonstrate, the irradiation of excitation by no means is restricted only to this zone, but, on the contrary, extends to the inhibitory points, 'washing out' the inhibition from them, as is shown by the disinhibition of the corresponding inhibitory reflexes. So what are we to believe? The law of generalization, which says that the irradiation of excitation spreads as far as the extent of the generalization, which in the case of differentiation of a given point from neighbouring points is very close? Or are we to believe the experimental data provided by Pietrova and Podkopayev, according to which, on the contrary, the irradiation of inhibition extends also to the inhibitory points? For if we accept this latter statement, it would follow that the application of a conditioned stimulus several times in succession should destroy the differentiation, since the excitation irradiating from the centre of this stimulus to the centres of inhibitory stimuli would allow these latter to reconnect with the centre of the unconditioned reflex. In such a situation no lasting differentiation could exist at all. But if we take the standpoint that in the case of differentiation of two proximate stimuli both processes, of excitation and inhibition, are concentrated (and even inductive relations exist between them), then how are we to interpret the results of Pietrova's and Podkopayev's experiments? It surely is unnecessary to add that it is not the facts that are in contradiction with one another, but their false interpretation.

To continue. From experiments on the disinhibition of inhibitory stimuli by weak extraneous stimuli, it results that the irradiation of excitation initiated by these stimuli involves the entire cerebral cortex, since the extraneous stimulus of one analyser is capable of disinhibiting the inhibitory stimulus of another analyser. From this can be inferred that if a weak stimulus becomes conditioned, the generalization from it should extend to all other stimuli, even those belonging to other analysers, while the conditioned stimuli of moderate strength, producing a concentrated excitation, should yield a very restricted generalization. This conclusion is false. There is no correlation whatever between the disinhibitory or inhibitory effect of extraneous stimuli on conditioned reflexes and the

degree of generalization of these stimuli when they become conditioned. These two categories of phenomena have simply nothing in common with each other, and a theory which unifies them cannot be correct. Moreover, it can be added that between weak stimuli, which disinhibit inhibitory reflexes, and stimuli of moderate strength, which inhibit active reflexes, there is no strict demarcation at all, such as should exist if we accept the theory above discussed. Most frequently the same stimulus which disinhibits an inhibitory reflex exerts an inhibiting effect (although feeble) on an active reflex So we should have to infer that an excitation caused by this stimulus is simultaneously irradiating and concentrating, which is obviously absurd.

In order to get over this contradiction, Pavlov accepted the explanation that an excitation irradiating from a centre of a weak foreign stimulus summates with the excitation of a point excited by a conditioned stimulus, with the result that top inhibition arises in this point.* But this explanation is wrong, since it would follow that an extraneous stimulus should inhibit only strong conditioned stimuli (such as evoke an excitation close to the top) but should increase the effect of weak conditioned stimuli But in reality it is rather the converse that occurs, since weak conditioned reflexes can be inhibited far more easily by extraneous stimuli than can strong ones. It is also worth noting that now, according to the Pavlov theory, one and the same fact, viz. external inhibition, is explained by two opposite cortical mechanisms (concentration of excitation and negative induction on the one hand, and irradiation of excitation and top inhibition on the other) and there exist no criteria at all to show which of these mechanisms is operating in any given instance.

6

Now we pass on to the properties of the process of inhibition. So far as the irradiation of this process is concerned, we must first and foremost point out that the results of the experiments which were conducted in order to demonstrate it by no means fulfil their role, i.e. they do not confirm the existence of irradia-

* Vide, for example, Pavlov, *Lectures*, vol. II, p. 90.

tion of inhibition at all. For, accepting the Pavlov conception, we should expect that after the application of an inhibitory stimulus the conditioned stimuli most approximated to it would be subject to inhibition first, and then those more remote, while the recession of the inhibition should occur in the converse order. The model for all this phenomenon should be a wave of inhibition spreading out from the inhibitory point, and then retreating back to it. But in reality, if we analyse the experiments which have been sufficiently precise and complete to enable definite conclusions to be drawn from them (such as the experiments of Anrep, Ivanov-Smolensky, Kreps and Podkopayev*), we find that inhibitory after-effect increases *simultaneously* for all the stimuli, and then simultaneously fades (cf. fig. 1). The difference between close and distant stimuli in relation to the inhibitory stimulus is simply that the first are inhibited (in the majority of cases, but by no means always) more deeply than the others, so that they reach zero point earlier and maintain it longer. It seems to us that the above-described actual course of inhibitory after-effect cannot be reconciled with the Pavlov conception, according to which the inhibition spreads from the point where it originates and returns to that point.

Further, as is evident from the experiments cited in the preceding chapter, the irradiation of inhibition occurs usually after the cessation of the inhibitory stimulus, but on its next application there is a momentary concentration of this process. This is founded on the fact that the conditioned stimulus applied concurrently with the inhibitory stimulus is anything but inhibited. Without discussing the point that it is difficult to imagine the mechanism of such a momentary concentration (if we consider that normally it occurs over several minutes), in the light of the fact described the phenomenon of the summation of inhibition arising on a repetition of the inhibitory stimulus is completely incomprehensible. For if with every application of such a stimulus the irradiating process of inhibi-

* Anrep, G W , *Russk fiz Zh.* 1917 vol. I, nos. 1–2 (Pavlov, *Conditioned Reflexes*, pp. 162 et seq). Ivanov-Smolensky, op. cit Kreps, E M., *Jubilee Volume* in honour of Pavlov's 75th birthday, 1925, p. 326 (Pavlov, *Conditioned Reflexes*, p. 205). Podkopayev, N. A., ibid. p. 298 (Pavlov, *Conditioned Reflexes*, p 209).

tion concentrates again, then every successive irradiation of inhibition begins from the beginning and cannot be summated with a process which has completely vanished. If we are to avoid the contradiction that arises here, we have to assume that the irradiation of inhibition and positive induction are completely independent processes, and can co-exist simultaneously in the same cells of the cerebral cortex. It has to be mentioned that Pavlov himself gave vague expression to this thought,* but this assumption cannot be reconciled with other statements of his.

Let us consider another fact. We remember from Chapter II (p. 11) that the conditioned inhibitor (i.e. the stimulus which jointly with the conditioned stimulus forms the inhibitory combination) attached to any other conditioned stimulus also causes an inhibition of its reaction. How are we to explain this fact? Every one of Pavlov's pupils will at once reply that this is a typical case of irradiation of inhibition. And yet we have just been saying that during the action of the inhibitory stimulus the process of inhibition is concentrated, and so, when we apply a conditioned stimulus together with the conditioned inhibitor, we should expect not a weakening, but an intensification of its effect But such a result *never* occurs. So we are bound to ask why the conditioned inhibitor breaks away from the general rule governing phenomena of internal inhibition, evoking an irradiation of this process without any concentration.

Further, it must be stated that positive induction is not at all the converse of negative induction, as Pavlov wished it to be. We remember that positive induction is based on the fact of an increase in the effect of active conditioned stimuli, applied during the action of inhibitory stimuli or immediately after them, whereas negative induction depends chiefly on the fact of the inhibition of the active conditioned reflexes by an extraneous stimulus. One sees easily enough that these two facts are by no means symmetrical, since in the case of positive induction the two stimuli (positive and inhibitory) are *homogeneous* conditioned stimuli (i.e. conditioned with the aid of the same unconditioned stimulus), whereas in the case of negative induction there is an interaction of two *heterogeneous* reflexes, e g. the orientation

* Pavlov, *Conditioned Reflexes*, lecture XII

reflex, evoked by the extraneous stimulus, and the conditioned alimentary reflex. If we wished to conduct experiments on negative induction analogous to those on positive induction, we would have to apply an inhibitory conditioned stimulus immediately after the active conditioned stimulus, and to show that the process of inhibition is then stronger than usual. But in reality we know from the Pietrova and Podkopayev experiments already cited that in these conditions, on the contrary, the inhibitory stimuli are disinhibited, which was taken to demonstrate the irradiation of excitation.

Furthermore, we must point out that the various facts on which the principle of negative induction is based have nothing in common with one another, and by no means justify ascribing them all to the same mechanism For instance, the phenomenon of inhibition of conditioned reflexes by extraneous stimuli on the one hand, and the phenomenon of the deepening of the inhibitory reflex after active conditioned reflexes (Krzyshkovsky's and Strogonov's experiments, p. 20) on the other, are facts of two completely different categories, occurring in completely different conditions, and the analogy between them is purely superficial. The first of these phenomena concerns heterogeneous reflexes, the other homogeneous reflexes, the first occurs in the course of action of a foreign stimulus or soon after, the other continues for a long time after the action of the conditioned stimulus; the first concerns active conditioned reflexes, which are inhibited; the other, on the contrary, has no influence whatever on such reflexes, and concerns only inhibitory reflexes, which are intensified. Could one imagine a greater heterogeneity between phenomena, or a more fine-drawn and artificial analogy between them?

We have the impression that in the course of discussion of the above experiments we have to some extent elucidated the secret of the already-mentioned flexibility of the Pavlov theory, and the ease with which it can be applied to explain the most widely different of experimental results. Indeed, once we are provided with the law of irradiation and concentration of excitation and inhibition, and the law of the mutual induction of these two processes, by arbitrarily applying these laws according to the

results obtained we can explain every fact concerning the interaction between stimuli. For every fact can be allocated to one of four categories: irradiation of inhibition, positive induction, irradiation of excitation, and negative induction. And whereas in Pavlov's hands the above-mentioned laws were none the less living and rich in content, and he continually attempted (though we think unsuccessfully) to find certain general principles governing the application of those laws, some of his pupils juggle arbitrarily with these concepts and quite mechanically stick corresponding labels on various categories of phenomena.

7

In concluding this survey we shall consider the phenomenon of sleep. As we said in Chapter II, this phenomenon is very frequently observed in association with internal inhibition, which led to the statement that in essence it is irradiation of inhibition. We would now like to adduce facts which contradict this statement.

First let us consider the oncoming of sleep during the monotonous repetition of an indifferent stimulus. According to Pavlov this fact is explained by the assumption that a stimulus reaching repetitively one and the same point of the cerebral cortex gives rise to an inhibition, owing to the extinction of the orientation reaction, and that this inhibition irradiates over the cortex and settles on subcortical centres * But if the investigators who performed these experiments† had carried out simple control experiments, leaving the dog at the stand without any stimuli, they would have discovered that sleep would come no less and possibly even more swiftly—a fact perfectly well known in everyday laboratory practice. Turning to our own life experience, we must observe that monotonous stimuli by no means necessarily help us to fall asleep, as the Pavlov theory supposes. For instance, in a brilliantly lit room it is unquestionably harder to fall asleep than in a darkened room, although continual illumination is a typical monotonous stimulus, to which our

* Pavlov, *Conditioned Reflexes*, chap. xv 'The problem of sleep', in the first volume of *Collected Works*, 1940, p 409, etc.

† Cf. Chechulin, S. I., *Arch Nauk biol.* 1923, vol. XXIII, nos 3–5, etc.

orientation reaction has long been extinct. Similarly, it is easier to fall asleep in perfect quiet than to the accompaniment of some monotonous sound. This is why if we want to sleep, we try to isolate ourselves from all external stimuli. Undoubtedly monotonous stimuli are less of an obstacle to our falling asleep than diversified stimuli, this being because of the adaptation of the organs receiving the given stimulus, and also because of the extinction of the orientation reaction to it. For this reason, after a certain time we can get accustomed to falling asleep even when comparatively strong external stimuli are in action (e.g. gunfire). But we have not the least reason to think that these stimuli help us to fall asleep, unless, perhaps, they have been conditioned to sleep by previous experience.

So it seems to us that Pavlov's statement that stimuli acting monotonously are *the cause* of sleep is quite without foundation and, indeed, is unsound. The most we can say is that stimuli of this kind *do not hinder* sleep, or hinder it only insignificantly But the best condition of falling asleep is the *absence* of stimuli, whether external or internal. And so in our view the prolonged sleep of an animal deprived of the majority of its receptors (cf. p. 24) is explained, in accordance with intuition, simply by the absence of external stimulation, and certainly not by an inhibition arising through the monotonous stimulation of the receptors still possessed by the animal.

Various experiments on conditioned reflexes also provide facts indicating that there is by no means the strict connexion the Pavlov theory assumes between internal inhibition and sleep. It was long since observed that if in experiments with conditioned reflexes only weak stimuli are applied (e.g. tactile, thermal, etc.), the dog grows more and more sleepy, and finally the experiments have to be broken off, because as soon as it finds itself on the stand it falls into a deep sleep. This even led Pavlov to assume that weak stimuli for some reason evoke a state of inhibition spreading through the cortex. But, on the other hand, we know that such stimuli evoke an irradiation of excitation, which is capable even of removing the state of inhibition from the inhibitory points. So once more we come up against a contradiction which seems to us inexplicable. Another fact is still more ex-

pressive. In a number of experiments performed in Pavlov's laboratory* it was shown that if the stimulation of a dog's skin with an electric shock is conditioned to food, it frequently leads to the animal falling asleep. Moreover, this phenomenon is so strongly marked that after a time the very process of attaching the electrode to the place usually stimulated causes sleep (by way of conditioning). This fact is quite incompatible with Pavlov's conception of sleep, and it cannot be explained from the viewpoint of that conception without artificially stretching the theory.

So, while not at all denying the wealth and interest of the facts concerning sleep discovered in the Pavlov laboratories, we must none the less state that the theory formulated on the basis of those facts is unsatisfactory, since some of the facts from this field are not explainable by it at all, while others it explains incorrectly.

It is hardly necessary to add that so far as we are concerned with the phenomena of sleep outside conditioned reflex experiments, namely, with experimental sleep evoked by stimulation of the hypothalamus, with sleep in encephalitis lethargica, or with natural or pathological sleep, these categories do not in the least require the Pavlov theory for their explanation, and, indeed, sometimes they are in obvious conflict with that theory.

8

It seems unnecessary to continue further with this discussion of the errors, internal contradictions and discrepancies to be found in the Pavlov theory. From what has been said, it is in our view quite clear that beneath the superficial harmony of this theory, and its apparent accordance with reality, there are many mutually contradictory statements which, moreover, do not accord with the general principles of functioning of the nervous system, generalizations which do not fit the facts, and arbitrary adaptation of experimental data to the requirements of the theory.

What is the reason for this? Can we even for one moment

* Vide, for example, Podkopayev, N. A., *Trudy Lab Pavlova*, 1932, vol. iv, Fiodorov, V. K., ibid. 1933, vol. v, p. 199, and others.

assume that the assertions of the Pavlov theory were formulated without adequate thought and without sufficient experimental bases? It is well known, especially to those who worked with him, how 'incessantly' Pavlov was absorbed in the problems raised in his laboratories, how much he thought about and tested each new conception, and how he hated pure speculation, unsupported by experimental evidence. We know, too, how many of the contradictions in his theory were observed by Pavlov himself, how frequently he spoke and wrote of them,* and how constantly he struggled, I do not hesitate to say struggled tragically, in order to eliminate them.

In our view the cause of this situation is clear. It derives from the circumstance that the main prerequisites and assumptions of the Pavlov theory are wrong, and so it is quite impossible to create an adequate and consistent theory of the cortical processes on their basis. Consequently the contradictions which we have pointed out, and many others we have not mentioned, are not the result of some oversight on the part of the creator of the science of conditioned reflexes, but are unavoidable within the framework of the theory he adopted, since they are inherent in its very fundamentals.

9

We would be guilty of an injustice if we maintained that Pavlov's views never aroused any doubts or reservations among his collaborators, and that they were immediately and uncritically

* We give a quotation from the last of his lectures devoted to the systematic presentation of the acquisitions in the realm of physiology of the higher nervous activity (Pavlov, *Conditioned Reflexes*, pp. 378–9) 'It is obvious that these special peculiarities of the research are, in many instances, the cause of fallacies, especially since it is so tempting to adhere to different fancied analogies and plausible generalizations—a tendency which cannot be too much guarded against in the present state of research The mind, so to speak, often fails to keep pace with the tremendous variety of interrelations, and this is why our interpretations were often too limited and led to errors which have had to be constantly corrected Indeed, I have no doubt that the presentation of the subject-matter attempted in these lectures will in the future still be corrected in many details Errors in interpretation, and errors sometimes in the methods of observations, are naturally to be expected in a study of such astounding complexity ' This passage faithfully reveals Pavlov's own doubts and hesitations concerning the correctness of the conceptions he put forward.

accepted by them. On the contrary, vital and sometimes stormy exchanges of opinion and ideas went on in his laboratories, both in the form of innumerable conversations, and also at the scientific meetings. Consequently, the statements that Pavlov formulated were usually the product of the collective mental effort of all the workers in the laboratories.

So it is not surprising that from time to time one or another of Pavlov's collaborators and pupils gave expression to views which were not in harmony with their master's, and which questioned certain, sometimes even essential, statements of his theory.

We shall devote this section to some of the more important and noteworthy criticisms of the Pavlov theory made by the more prominent of his followers.

In 1932, as the result of experiments he had made, Anokhin decided that Pavlov's statement that the seat of the process of internal inhibition is the centre of the conditioned stimulus was erroneous.* He based his criticism on the fact, established in his laboratory, that in the process of internal inhibition a diminution or disappearance of the proper conditioned response (e g. salivation) is by no means accompanied by a weakening of other reactions of the organism. On the contrary, there is an intensification of breathing activity, as also of some motor reactions. Hence it was inferred that the inhibitory process cannot occur in the centre of the conditioned stimulus, since in that case *all* the animal's reactions would be bound to suffer inhibition.

Anokhin's collaborator, Balakin, who drew attention to this fact, wrote about it as follows:† 'All conditioned stimuli falling on any part of the animal's sensory surface always and in all conditions evoke in the receptory points of the cerebral cortex a process of excitation; but the absence of, let us say, the secretory indicator may be due to an absence of excitation in the executive part of the reaction, i.e. either in the peripheral secretory centre, or in the "cortical representation" [Pavlov's phrase] of the corresponding organ.'

* Anokhin, P. K., *Russk fiz Zh* 1933, vol xvi, no 5.

† Balakin, S , in *Problems of the Centre and the Periphery in the Physiology of Nervous Activity*, collection of papers, etc., edited by Anokhin, 1935, p. 379.

Thus long ago Anokhin and his collaborators found a weakness in the Pavlov theory and criticized it in a statement with which we wholeheartedly agree. But did Anokhin draw the conclusions that follow from his criticism? The fact of motor excitation during the action of the internal inhibitor was perfectly well known to Pavlov, yet it did not cause him to modify his view. Anokhin, who, on the basis of facts he had observed, undermined one of the fundamental assertions of the Pavlov theory, did not even attempt to show how otherwise the facts on which it was based ought to be explained. It is clear that if this statement is rejected the principles of irradiation, positive induction and many other laws of the functioning of the cerebral cortex laid down by Pavlov must also be rejected. But in that case we are bound to ask what is to replace them, and Anokhin does not answer this at all. He was undoubtedly quite aware what an enormous mass of factual material this assertion was based upon and how long Pavlov had hesitated and wavered before he finally adopted it. So either we must find a different explanation for all this material, or the facts to which Anokhin drew attention must be explained in harmony with the Pavlov theory. This second alternative is by no means difficult, for we need only accept the view (which was adopted by Pavlov himself in regard to motor excitation) that the excitation of the breathing centre is due to positive induction caused by the process of inhibition concentrated in the centre of the inhibitory stimuli. This must not be taken as meaning that we agree with this explanation; we simply wish to show that the facts under consideration do not destroy Pavlov's assertion, and that, if we accept his theory as a whole, we must take account of this particular explanation.

Similarly, we cannot agree with the criticism, or rather modification, of Pavlov's views, which Asratian attempted to make in one of his works.* This author reveals a correct tendency to base the laws governing the higher nervous activity on the general laws of the functioning of the nervous system. From this standpoint the author attempts to explain the law of the irradiation and concentration of inhibition in a manner

* *C.R. Acad. Sci. U.R.S.S.* 1934, p 146.

more intelligible to modern physiology. He claims that inhibition initiated in a particular point of the cerebral cortex does not spread as such, but that the cells transfer to one another a state analogous to Sherrington's 'central inhibitory state'. The concentration of inhibition is not a recession of this process to its starting point, but simply its gradual disappearance. The author adopts an analogous theory in regard to the irradiation and concentration of excitation.

We must point out that this correction to the Pavlov theory in no way saves the situation in regard to all the objections we have made in preceding sections. Moreover, the author did not test out whether it can be maintained in face of all the known facts. It is easy to show that it cannot. For if the irradiation of excitation concerned not the process itself, but only the sub-threshold excitatory state, it would be impossible for cells in such a state to enter into connexion with the centre of the unconditioned stimulus, i e. generalization of conditioned reflexes would be impossible.

Finally, we will consider certain views of another outstanding investigator in this field, Kupalov.

Kupalov,* like Anokhin, also pays much attention to the problem of the seat of the process of inhibition, and extensively discusses all the possibilities that may be considered. He adduces the fact (known for a long time past) that, if the inhibitory stimulus is suddenly reinforced by an unconditioned stimulus, the unconditioned reflex is inhibited. Where does this inhibition occur? After considering various possibilities, he comes to the conclusion that it could occur only in the 'cortical representation' of the unconditioned centre, and consequently he declares: 'our investigations have shown that in all inhibitory reflexes the inhibition is addressed not so much to those nerve elements associated with the reception of the conditioned stimulus, as to further elements, which take part in the long-protracted course of the reaction. To express the position in spatial terms, this inhibition is localized not in the centre of the conditioned stimulus, not in the receptory part of the cortex,

* Vide Kupalov, P S, *Arch Nauk biol* 1939, vol. LIV, p 5, ibid. 1939, vol. LIV, p. 14.

or not only in it, but, in the case of alimentary reaction, also in the cortical alimentary centre.' As we see, after some hesitation Kupalov is disposed, like Anokhin, to accept that internal inhibition takes place in the centre of the unconditioned stimulus, but he, too, does not draw the logical consequences from this statement, and in other cases operates entirely within the limits of the Pavlov theory, paying no attention to the contradictions that arise. It is worth recalling here that Pavlov ascribed the inhibition of the alimentary reflex under the influence of an inhibitory stimulus simply to the irradiation of inhibition into the alimentary centre. We must admit that if Pavlov's theory is to be accepted it is difficult to think of any other satisfactory interpretation of this phenomenon.

Summarizing what we have said, we consider that the views expressed by Anokhin, Kupalov and others are undoubtedly due to their observation of defects in the Pavlov theory, but that they were not prepared to carry their criticism to its logical consequence, and so it could not avoid being fragmentary and incomplete in its nature.

ADDENDUM

The Beritoff Theory

We think we are right in saying that the first to draw attention to the discrepancy between the present-day physiology of the central nervous system and Pavlov's theory was the eminent Georgian physiologist, I. S. Beritoff. In his monograph entitled *Individually Acquired Activity of the Central Nervous System* (Tiflis, 1932)* he subjected Pavlov's theory to severe and ruthless criticism, pointing out its entire incompatibility with the evidence of modern physiology. Further, on the basis of experimental material accumulated by the Pavlovian school, and also of his own experiments, he attempted to establish his own theory of higher nervous activity, based on the general physiology of the central nervous system.

So far as Beritoff's criticism of the Pavlov theory is con-

* See also Beritoff, I S., *Brain*, 1927, vol. LXXVII, pp. 109, 358.

cerned, we share his views almost in their entirety. His fundamental thesis, that the Pavlov school developed its work in almost complete isolation from other workers in the field of neurophysiology and made very little use of their achievements, is undoubtedly sound, and is recognized as such to-day even by pupils of Pavlov himself.* The examples he adduces of the discrepancies in this field are wholly convincing, and largely coincide with the arguments as presented in § 3 above. However, so far as the positive aspect of Beritoff's work is concerned, i.e. his own theory of higher nervous activity, we think it is unsatisfactory.

The basic assumption of Beritoff's theory is his law of the 'interdependent irradiation of excitation'. According to this law, excitation arising in a certain zone of the cerebral cortex irradiates over the nervous tissue in all directions, the strength of this irradiation along every nerve path being dependent on the excitability of this path or the centre to which it leads and the excitability of all the other centres or nerve paths originating from this zone. The more excitable the given path, the stronger is its excitation, and the weaker is the excitation of other nerve paths. As Beritoff himself emphasizes, nervous excitations behave like an electric current running along a ramified conductor. He assumes this property of 'interdependent irradiation' to be specific to the cerebral cortex, and explains it by the distribution of bio-electric currents with varying strength in varying directions, assuming that these currents intensify the excitability of the corresponding nerve paths, the degree of excitability increasing with the strength of the currents.

If two centres of the cerebral cortex (say the centre of an indifferent stimulus and the centre of an unconditioned stimulus) are excited simultaneously, nervous impulses are propagated from these centres in all directions, but the strongest stream of impulses runs along the paths connecting these two centres. This is because the excitability of the unconditioned centre during its excitation is very high and so, according to the law of interdependent irradiation, it accumulates the greater part of the nerve impulses, to the detriment of other paths. With

* Vide Kupalov, P S , *Arch. Nauk biol.* 1938, vol. LIV, p. 14.

repetitive concurrence of these two excitations the paths connecting them, being the most active, grow more and more excitable, and therefore they more and more concentrate in themselves the entire traffic of nerve impulses propagated from the centre of the indifferent stimulus. As a result the conditioned (or individually acquired, as Beritoff calls it) reflex is formed, since the centre of the indifferent stimulus being excited sends impulses predominantly in the direction of the unconditioned centre.

As to the irradiation of excitation from the centre of the unconditioned stimulus, it is rather insignificant, since the main part of the impulses is conducted to the executive organs. But when the conditioned stimulus ceases to be reinforced the excitation of the centre of the unconditioned stimulus dissipates in all directions, and predominantly in the direction of the centre of the conditioned stimulus, this last centre being in a state of high excitability. With the repetitions of unreinforced trials such retrogressive connexions grow more and more excitable, and they gather to themselves all the excitation originated in the centre of the unconditioned stimulus, to the detriment of other directions. In consequence the conditioned response gradually diminishes, and eventually subsides altogether. So the entire body of phenomena related by Pavlov to the concept of internal inhibition is treated by Beritoff not as inhibition, but as the result of establishing retrogressive connexions between the corresponding centres To provide 'direct' proof of this assumption Beritoff cites an observation by Podkopaev,* who found that the rhythmic tactile stimulation of a paw began to evoke the defensive flexion of the leg, since it became a differential inhibitor. In this case, argues Beritoff, the excitation transferred from the centre of the conditioned to the centre of the unconditioned stimulus afterwards runs back to the centre of the conditioned stimulus, summates with the original excitation of this centre, and in this manner evidently becomes so strong that it forms the source of a defensive reaction.

The inadequacy of this interpretation can be demonstrated without difficulty. For the fact just cited occurs only in the

* Podkopaev, N A., *Trudy Lab. Pavlova*, 1926, vol. 1/2–3, p. 187.

case of alimentary reflexes; where acid (or other defensive) conditioned reflexes are concerned we have clear evidence which points to the direct opposite. For instance, experiments by Petrova* showed that the defensive flexion to the tactile stimulation of a paw was obtained when this stimulation was reinforced by acid (and not differentiated as in Podkopaev's experiments), and so no conditions for the formation of retrogressive connexions were present. That it was really so can be demonstrated by the fact that the proper conditioned reaction to this stimulus (i.e. salivation) was by no means impeded, which makes the assumption that retrogressive connexions were formed inadmissible.

Still more convincing proof of the inadequacy of Beritoff's conception can be found in Fiodorov's experimental results † This author established an acid conditioned reflex to a weak electrical stimulation of the dog's skin, with the result that the local defensive reaction against the electrical shock was greatly enhanced, but simultaneously this stimulus evoked a powerful acid conditioned reaction. These two reactions by no means neutralized each other (as they should if the laws of interdependent irradiation and retrogressive connexions were valid), rather the contrary, mutual facilitation was observed. The local defensive reaction to the electric shock was only suppressed when acid reinforcement was replaced by alimentary reinforcement.

We are confronted with a similar situation if we take into consideration the experiments of Bezbokaya, which were described in the foregoing chapter (§ 7). This author found that the aggressive reaction in a dog greatly enhanced the alimentary conditioned reflexes. Now, if the centre of aggression, being excited, drew off to itself the excitation which was directed to the alimentary centre, the alimentary reflex should be reduced. The same should occur if the aggression produced a general excitation of the cerebral cortex, which, according to the law of interdependent irradiation, would cause a dispersal of the nervous impulses in all directions. But any increase in alimentary

* Pietrova, M , Diss , Petersburg, 1917

† Fiodorov, V K., *Trudy Lab. Pavlova,* 1933, vol. v, p 199

reflexes under the influence of aggression is, from the viewpoint of the Beritoff theory, quite incomprehensible.

All the above-cited facts are in opposition both to the conception of interdependent irradiation of excitation and to the formation of retrogressive connexions between the centres when the conditioned stimulus is non-reinforced. And, as will be shown in Chapter VII, these facts can be satisfactorily explained on the basis of general neurophysiological principles without the necessity of applying any additional mechanisms

More recently Beritoff has supplemented his theory on the basis of his conception of the significance of neurophil in the processes of inhibition.* These supplements not only did not lead to any clarification of his views, but, on the contrary, made them more confused For he now ascribes a dual mechanism to one and the same phenomenon: there is the mechanism which he formerly accepted, and the mechanism associated with inhibition caused through the action of neurophil.

There are other difficulties of interpretation in Beritoff's theory which could be pointed out. But we think it unnecessary. This theory has played no great part in the development of the physiology of higher nervous activity, and, unfortunately, has not even induced workers in the Pavlov school to revise their views and to look for other interpretations of the facts obtained in this field. The only real service Beritoff has rendered, in our view, is that he was the first to draw attention to the gulf that exists between the physiologies of the higher and lower nervous activity, and was the first to attempt to bridge this gulf.

* Beritoff, *Trans J Beritashvili Physiol Inst.* 1937, vol. III, 21; *Communication of the Georgian Filial of the Soviet Academy of Sciences*, 1940, vol I, 149.

Chapter IV

General laws of the functioning of the nervous system

1

We give the following brief outline of the laws governing the functioning of the nervous system for two reasons. First, we desire to make our work intelligible to those readers who are not fully acquainted with these laws, and who, therefore, might otherwise not thoroughly understand our further argument. Secondly, despite the great progress of recent years in the physiology of the central nervous system, many problems in this field remain unclarified, and there is not as yet unanimity of thought in regard to them. The necessity to take into consideration the great mass of new facts which have been provided by the physiology of higher nervous activity, and the task of incorporating them in the general aggregation of neurophysiological data oblige us, among various conceptions, to choose those which will serve for explanation of these new facts, and to reject others with which they could not be reconciled

So the present exposition is entirely subordinated to the ends it is intended to serve, i e. it discusses only those laws of nervous activity which will be of use to us later and, without raising any controversies, presents only those hypotheses which we shall use for the construction of a theory of cortical activity.

Apart from this, one other circumstance has governed the character of the present exposition. In the physiology of higher nervous activity we do not deal with such stimuli as single volleys of impulses, and with such reflexes as act through two- or even three-neuron reflex arcs. The stimuli which we make use of in this field are lasting and compound, the reflex arcs are extensively ramified and multi-neuron in nature. Consequently, in order to get an idea of the phenomena which are found in this field of neurophysiology, one needs to make a certain extrapolation of the laws discovered on relatively simple reflex preparations to phenomena incomparably more complex. An

attempt to give such an extrapolation will also be made in this chapter.

It is perhaps unnecessary to emphasize that for the laws and hypotheses on which we base our exposition we are chiefly indebted to Sherrington and his school, and that, despite the modifications that have been introduced in the subject of recent times, as a whole his conception remains valid for to-day, and serves as the foundation of the work which we have undertaken in this book.

2

A nervous impulse, conducted along the axon of one nerve cell to the body or dendrites of the next cell, causes either a state of excitation, or a state of inhibition in the spot at which it arrives (i.e. at the synapse). It will be to the point to consider these two states separately.

The essential point in our understanding of the functioning of nerve centres was the realization that a single centripetal (pre-synaptic) nervous impulse, reaching the nerve cell and exciting it, in normal conditions is insufficient to cause a discharge of the centrifugal (post-synaptic) impulse.* The excitation which one afferent impulse produces in a nerve cell is subliminal and must summate with excitations caused by other afferent impulses for the cell to be excited above the threshold. This gave rise to the concept of the 'central excitatory state', i.e. a state which is caused by nervous impulses arriving at the nerve cell and which, having accumulated in sufficient amount, produces a discharge of the efferent impulse, but, not reaching the threshold, is gradually dissipated. If the intimate nature of excitation is regarded as consisting in a depolarization of cell membrane, then the subthreshold 'central excitatory state' will be due to its partial, incomplete depolarization.

As to the precise mechanism of the initiation of post-synaptic impulses in the nerve cell two different views can here be taken into consideration.

* Later in the text, when using the terms 'centripetal (or afferent) impulses' and 'centrifugal (or efferent) impulses' we shall relate them only to a given centre, and not necessarily to the entire central nervous system.

According to Lorente de Nó* a nervous impulse arriving at the nerve cell produces at its synapses a 'synaptic excitatory process'; this state differs from the Sherringtonian 'central excitatory state' in being purely local and lasting a much briefer period, only the fraction of a millisecond (as compared with the dozen or so milliseconds' duration of the central excitatory state). In itself it is inadequate to excite the neuron above threshold, for this to occur, it must summate with similar processes brought about in *neighbouring synapses.* As it is of such brief duration, this summation can occur only if there is an almost simultaneous arrival of various impulses in the given discrete zone of the cell surface.

Lorente de Nó has shown that every axon ramifying around the nerve cell possesses synapses scattered over that cell's body, but on any small zone of its surface only synapses originating from different axons are to be found. It follows that the liminal excitation of a nerve cell is always initiated by the simultaneous action of impulses originating from different axons, i.e it is the result of the *spatial summation* of impulses

On the other hand, as was stated recently by Eccles,† impulses reaching the nerve cell evoke on its surface a prolonged and widespread 'synaptic potential' of catelectrotonic origin. Eccles assumes that this 'synaptic potential', if sufficiently strong, is the actual cause of neuron discharge. Since it lasts more than 10 milliseconds it can easily summate with 'synaptic potentials' evoked by successive volleys of impulses, and thus the *temporal summation* of afferent impulses comes into being. Lloyd in his recent paper‡ confirmed the existence of prolonged facilitation evoked by a single volley of afferent impulses reaching the motoneurons, and called it 'residual facilitation'.

It is difficult to foresee now which of these mechanisms will prove to be true and responsible for the neuron discharge It is possible, as is assumed by Lloyd (*op. cit.*), that they both share in

* Lorente de Nó, R., *Amer J. Physiol* 1935, vol CXI, p. 272, ibid p 283; ibid vol CXII, p 595; ibid. vol CXIII, p 505; ibid p 524; ibid. 1938, vol. CXXIII, p 388 (with H. T. Graham), *J Neurophysiol* 1938, vol I, p. 187; ibid. p 195, ibid. p 207; ibid 1939, vol II, p 403

† Eccles, J C., *J. Neurophysiol* 1946, vol IX, p 87.

‡ Lloyd, D. P. C, *J Neurophysiol* 1946, vol IX, p. 421

synaptic transmission, one being the immediate cause of the discharge of the neuron and the other increasing its excitability. In any case it has to be noted that the above described controversy does not affect the problems dealt with in the present book, for they can be analysed in the terms of either conception.

The structure of the great majority of reflex arcs is such that they include two fundamental types of circuit.* multiple chains of neurons (the type M) and closed chains of neurons (the type C) (fig. 2). The multiple chains cause a considerable temporal dispersion of the impulses falling on the nerve cell, and enable the summation of a number of volleys, sent one after another at quite wide intervals of time, since direct impulses set up by succeeding stimuli can arrive at this cell simultaneously with impulses set up by preceding stimuli, but reaching it through many internuncial relays. Closed (or self-re-exciting) chains are responsible for the fact that the reflex response lasts much longer than the stimulus itself and subsides very slowly, since in these conditions every cell-discharge becomes the source of its renewed excitation. And so such closed chains of neurons, once they have been excited, can go on discharging 'spontaneously' for a long period, until an end is put to their activity by some additional stimulus, which has an inhibiting effect on one of the links in this circuit and so stops all this spontaneously acting mechanism (Compare, for example, the effect of an inhibitory stimulus on the after-discharge of a crossed extensor reflex.†)

Taking into account the manner in which the nervous impulses act on the cell which they reach, and the above described arrangement of neurons in nervous centres, we can represent the reflex activity in the nervous system as follows. Each afferent impulse, as the result of the numerous ramifications of the fibre along which it runs, reaches many neurons, and, owing to the numerous ramifications of axon terminals around every single cell, as well as owing to the lateral chains of the type M, it arrives

* Ranson, S W. and Hinsey, J C, *Amer. J. Physiol.* 1930, vol xciv, p 471 Lorente de Nó, R, *Arch Neurol. Psych* 1933, vol xxx, p 245.

† Liddell, E G. T and Sherrington, C. S., *Proc Roy Soc.* B, 1925, vol. xcvii, p. 488.

at the nerve cell in the form of hundreds of both synchronized and successive impulses. If we take into account the fact that a normal stimulus sets up not one impulse, but a long series of closely consecutive impulses in every afferent fibre, and that many afferent fibres contact with each neuron, we see that as

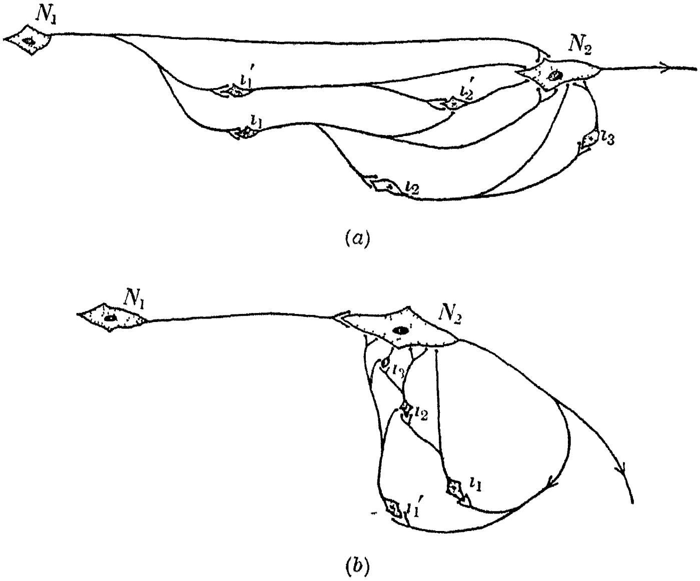


Fig. 2. Chain arrangement of neurons according to Lorente de Nó. (*a*) The multiple chain (M) The neuron N_2 is excited by the neuron N_1 either directly, or indirectly by the intermediary of various chains of internuncial neurons: i_1', i_1'–i_2', i_1–i_2–i_3, etc In this manner the single impulse set up by the neuron N_1 is transformed into numerous impulses reaching the neuron N_2 with temporal dispersion. (*b*) The closed chain (C) The neuron N_2 excited by the neuron N_1 re-excites itself through the intermedial neurons i_1, i_1', i_2, i_3. If this re-excitation is strong enough to produce a repeated discharge of the neuron N_2, the activity of this neuron may last a long time without any influx of impulses from the neuron N_1.

the result of stimulation an abundant and prolonged shower of impulses falls on the neuron. In view of the Lorente de Nó conception we have just presented we must conclude that the origination of an efferent impulse in a neuron is a matter of statistics. If the shower of impulses falling on a neuron is weak,

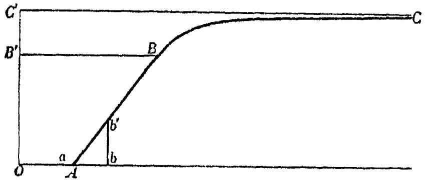
then it is very unlikely that so many of these impulses will assemble simultaneously on a small zone of its surface as will cause depolarization adequate to ensure the discharge of a new impulse. Therefore the cell will be in a state of permanent subliminal excitation, which, in accordance with its relative strength, will occasionally discharge in the form of an efferent impulse. But if a 'downpour' of nervous impulses falls on a nerve cell, there will be a very great probability of threshold depolarization arising on one or another zone of its surface, and the cell will discharge with great frequency, limited only by its subnormality periods.

3

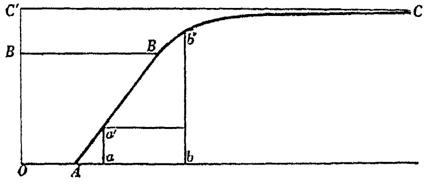
As is seen from the foregoing discussion, the frequency of the efferent impulses set up by the nerve cell is in strict relation to the density of afferent impulses falling on it If we wish to express this relation graphically the curve shaped approximately as in fig. 3 will be obtained. This curve is constructed according to the following assumptions: (1) When the density of the afferent impulses is small (less than *OA* in the diagram) they are insufficient to initiate the discharge of the cell and they hold it in a state of subliminal excitation. (2) When the density of afferent impulses rises above the threshold (i.e. it becomes greater than *OA*) a series of efferent impulses is set up by the neuron, the rate of this series being, within limits, more or less proportional to the density of the incoming impulses. (3) When the frequency of the efferent impulses achieves a definite value (*OB'* in the diagram) they begin to follow one another in a period of subnormality (which period, it should be recalled, is the more protracted, the greater the frequency of discharge),* and in consequence their further increase is slowed down: the curve bends towards the horizontal. The theoretical asymptote of the curve is determined by the length of the absolute refractory phase of the neuron.

If we are concerned not with the frequency of efferent impulses but with the magnitude of the reflex response (as is the case in the study of conditioned reflexes) then the height *OB'*, at which the curve *ABC* bends, is less than in the previous case,

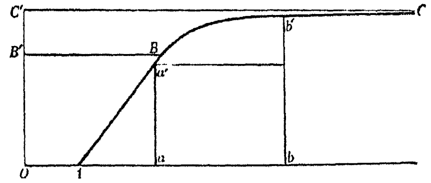
* Lorente de Nó, R , *Amer. J. Physiol.* 1938, vol. cxxiii, p. 388.



(a)



(b)



(c)

Fig 3. The characteristic of the functional system and the principles of summation of allied reflexes Abscissae: the density of the shower of excitatory impulses falling on the neuron. Ordinates: the size of reflex response of the given effector-unit (*a*) Summation of two simultaneous subliminal stimulations *Oa*, the density of the incoming impulse shower set up by each stimulus acting independently, $Ob=2Oa$, their concurrent action; *bb'*, the size of the corresponding reflex response. (*b*) Summation of two simultaneous supraliminal weak stimulations. *Oa*, the density of the incoming impulse shower produced by each stimulus, *aa'*, the corresponding reflex response; $Ob=2Oa$, the density of impulse shower caused by two stimuli acting concurrently, *bb'*, the corresponding reflex response. The reflex response to the joint stimulation exceeds the sum of the responses to each stimulation separately ($bb'>2aa'$). (*c*) Summation of two simultaneous strong stimulations. The symbols as in (*b*); the reflex response to the joint stimulation is less than the sum of the responses to each stimulation applied separately ($bb'<2aa'$).

since the maximal response of the effector organ is reached at a rate of efferent impulses usually much smaller than that allowed by the efficiency of the discharging neuron.* Other features of the curve will remain without change.

We shall call the curve *ABC* 'the characteristic of the neuron', or 'the characteristic of a functional system' if the magnitude of the response and not the rate of discharge is taken into consideration.

Suppose now that two independent showers of excitatory impulses fall on the given nerve cell. The results of their summation are also represented in fig. 3.

If the density of each shower is such that it produces only subliminal excitation of the cell, their concurrent action may be quite sufficient to evoke supraliminal excitation and to generate a series of efferent impulses of a greater or lesser rate Then we have the phenomenon of *facilitation* of two subliminal stimulations (fig 3*a*).

If two stimuli set up separately evoke a weak supraliminal excitation of the nerve cell, then their concurrent action may also result in facilitation, the conjoint reflex response more or less exceeding the arithmetical sum of both separate responses (fig. 3*b*).

But the situation changes when the stimuli become stronger, i.e. the arithmetical sum of their effects exceeds the height *OB′*. Then, instead of facilitation, *occlusion* takes place, a phenomenon which is due to the existence of the maximal response of the given effector unit. As fig. 3*c* shows, its result is that the joint effect of two strong stimuli is much less than the sum of their separate effects. Occlusion is the more manifest the stronger are the two acting stimuli, or at least one of them In the case of one of them being maximal, i.e. of its giving a maximal response of the functional system, occlusion is complete: this means that the conjoint effect of the two showers of afferent impulses does not exceed the effect of the stronger one acting separately.

Before we proceed we should like to draw attention to some

* Adrian, E D. and Bronk, D. W., *J. Physiol* 1929, vol LXVII, p 119, Sherrington, C. S., *Proc Roy Soc.* B, 1929, vol. CV, p. 332, Cooper, S. and Denny Brown, D., ibid. p. 363.

terminological vagueness concerning the concept 'excitation', which emerges from our present discussion.

It is not difficult to observe that this term has been used so far in two slightly different meanings:

(1) The excitation of a neuron (or centre) signifies that it is stimulated by excitatory impulses, and the measure of the excitation *thus* conceived is the density of the afferent impulses reaching the neuron.

(2) But excitation of a neuron or centre also means that it is thrown into activity, i.e. that it is discharging efferent impulses, and the measure of the excitation *thus* conceived is the frequency with which the efferent impulses are fired off.

Very frequently there is no need whatever for distinction between these terms, since excitation in the second sense is completely dependent on excitation in the first sense, so it makes no difference which of these meanings is intended. But this is far from always the case. If, for instance, very many excitatory impulses bombard a given centre, but for some reason or other its excitability is diminished, then its excitation in the first sense is considerable, but its excitation in the second sense is small, since, because of its momentary properties, the centre is discharging at a slower rate. A similar situation arises when, in addition to excitatory impulses, inhibitory impulses fall on the centre. Then it is again strongly excited in the first sense, but its excitation in the second sense may be insignificant, since the inhibiting impulses have a hindering effect (see below). The converse phenomenon occurs when the excitability of the given centre is increased (e g. under the influence of strychnine). Then a weak excitation of this centre (in the first sense) is sufficient to bring about a considerable excitation (in the second sense).

In order to avoid misunderstanding, wherever it is necessary to distinguish between the two meanings we shall use the term *active excitation* or *activation* of the nerve cells, whenever the stress is laid on the second meaning, i.e. on the amount of the activity produced by the centre. In cases where it is not essential to distinguish the two concepts and where the term 'excitation' simultaneously implies both meanings, the adjective 'active' will be dropped.

4

We pass now to consideration of the mechanism of central inhibition As we know, the problem of the intimate nature of inhibition is not yet resolved, and, therefore, in our interpretation of this process we must resort more often to incompletely substantiated hypotheses than was the case in our discussion of the process of excitation.

Of course we have no intention of dealing with the problem of inhibition in all its extent. We have only to remember that while, chiefly because of the works of Gasser and Lorente de Nó,* for some years before the war there was a strong tendency to accept the monistic theory of excitation and inhibition (according to which there are no separate 'inhibitory synapses', and inhibition can be entirely accounted for as the result of a subnormal phase after the passage of nervous impulses in sufficiently rapid succession) later works by Lloyd and Renshaw† have clearly shown that this explanation does not cover all the cases of inhibition, and that beside the 'indirect inhibition' due to the subnormal phase of a discharged neuron one must admit the existence of 'direct inhibition' caused by inhibiting impulses.

Taking into account the evidence provided by the physiology of higher nervous activity, we have to accept the fact that 'direct' inhibition unquestionably exists, and that it is this type which plays the main, if not the exclusive role in all inhibition phenomena found in conditioned reflex activity. Moreover, it seems to us that, if our interpretation of this activity is correct, it provides additional support for the dualistic theory of excitation and inhibition. And so, without considering whether 'indirect inhibition' plays any role in the normal functioning of the nervous system,‡ we shall discuss only the conception of inhibition as a process separate from excitation.

* Vide, for example, Gasser, H. S., *Bull N.Y. Acad Med* (Harvey Lecture), 1937, vol. XIII, p 324, Lorente de Nó, R., *J. Neurophysiol.* 1938, vol I, p 207.

† Lloyd, D. P. C, *J. Neurophysiol* 1941, vol IV, p. 184, ibid 1943, vol VI, p. 111, ibid. 1946, vol. IX, p 421; ibid. p 439. Renshaw, B, ibid. 1941, vol. IV, p. 167; ibid. 1942, vol V, p. 477; *Amer. J. Physiol.* 1946, vol. CXLVI, p. 443.

‡ Even if the answer is in the affirmative, we consider that the mechanism

So it is assumed that excitatory and inhibitory impulses (i.e. impulses exerting excitatory or inhibitory effect on the nerve cell they reach) are conveyed through different pathways, though where the actual difference between them lies—whether in various types of nerve terminals, or in their various localization, or else in various kinds of neurons which set up impulses—is as yet unknown. It is also not clear whether one and the same axon, when ramifying, can give different types of terminals to different nerve cells, or whether it may convey impulses of only one character. In our further discussion we deem it prudent to operate with the second assumption.*

As to the time course of inhibitory action exerted by inhibiting impulses on a nerve cell, the first clear answer to that question was given recently by Lloyd† who has shown that it is more or less constant and lasts as long as 'residual facilitation', i.e. about 10 milliseconds. Thus, if certain inhibitory reflexes possess longer and others shorter after-effect, if some of them have a greater and others a lesser latency, if certain reflexes 'open' at once and others gradually, showing recruitment, these variations depend exclusively on the structure of their reflex arcs, i.e. on the same factors that determine the course of excitatory reflexes.

From this it follows that if a nerve cell is subjected to the simultaneous action of excitation and inhibition, showers of excitatory and inhibitory impulses fall on it, and the activity of the cell depends on their respective density If, for instance, at a given moment sufficient excitatory impulses arrive at the cell membrane to initiate a discharge in ordinary conditions,

of 'direct inhibition' is so different from that of 'indirect inhibition', that it seems improper to give the common name of 'inhibition' to both these phenomena. So we propose to retain this term for the phenomenon of 'direct inhibition', which after all is nothing else than the well-known Sherringtonian 'antagonistic inhibition', and to introduce the term 'subnormality depression' for 'indirect inhibition'.

* From the experiments conducted by Kato and his collaborators (Kato, G , Kaku, Z. and Tasaki, I., volume dedicated to Professor Berytasvili, Tbilisi, 1936, p. 203), it follows that one and the same afferent nerve fibre is capable of carrying excitation to certain neurons and inhibition to other neurons But this fact does not contradict the thesis we have adopted, for it can be assumed that the inhibition of a given group of motoneurons was transmitted to them not directly, but through internuncial neurons.

† Lloyd, D. P. C., *J. Neurophysiol.* 1946, vol. ix, p. 421.

but simultaneously inhibitory impulses fall on it, then the excitatory impulses may prove to be inadequate, and the neuron will fail to discharge at that moment. If the inhibitory impulses are much more frequent than the excitatory ones, the cell's activity will be completely suppressed; if the predominance of the inhibitory impulses is less, they will only slacken the rate of discharge to a greater or lesser degree; if, on the other hand, the inhibition is not so very strong, whereas more excitatory impulses fall on the cell than is necessary to produce the maximal reflex response, then the inhibitory stimulus may have no influence whatever on the course of the reflex, etc.*

From what has been said it is seen that, without in the least prejudging the intimate nature of inhibition, we may assume that inhibitory impulses *heighten the threshold of excitability* of the nerve cell, since under their action more excitatory impulses than the normal are required in order to initiate a discharge of an efferent impulse in the neuron This means that the curve of characteristic of nerve cells represented in fig. 3, under the influence of inhibitory impulses, will be shifted to the right, the extent of this shift giving the measure of the density of inhibitory impulses arriving at the cell. As for the shape of the curve, it remains unchanged, since the discharge mechanism of the neuron itself is in no way affected (fig. 4).

As is seen in fig. 4, if the shower of excitatory impulses falling on the cell is very heavy, its effect is hardly affected at all by moderate inhibitory influence, if it is not so very dense, inhibitory impulses diminish the frequency of the reflex discharge, if inhibitory impulses are abundant the excitation of the cell becomes subliminal, etc. In other words, all the relations obtained by the concurrent action of excitatory and inhibitory impulses can be easily represented in our diagram.

5

Having discussed the action of excitation and inhibition upon the single nerve cell, we shall now consider the manner in which these processes interact throughout the nervous system. Long

* The reader will find many demonstrative examples of such relations in Sherrington's Ferrier Lecture. *Proc. Roy. Soc* B, 1929, vol cv, p. 332.

since, Sherrington drew attention to the circumstance that the reflex we operate with in experimental work is 'a convenient if not a probable fiction', and that in reality the organism's response to a stimulus is always compound and involves a large number of effectors. Thus every reflex throws the whole of the nervous system into activity, causing excitation in certain elements, inhibition in others, and alternating states of excitation and inhibition in yet other elements. And so particular reflexes

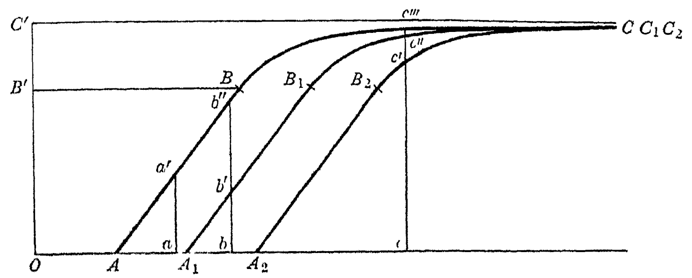


Fig 4. The influence of inhibition upon the characteristic of the functional system. Abscissa: the density of the shower of excitatory impulses falling on the neuron Ordinate the size of the reflex response of the given effector-unit ABC, 'normal' characteristic of neuron. $A_1B_1C_1$ and $A_2B_2C_2$, the characteristics of neuron bombarded by lesser or greater quantity of inhibitory impulses *Oa*, weak excitatory stimulation of the neuron. *Ob*, moderate excitatory stimulation of the neuron *Oc*, strong excitatory stimulation of the neuron The shift of the curve ABC to the position $A_1B_1C_1$ causes the response to the stimulus *Oa* to become subliminal, the response to the stimulus *Ob* to become greatly diminished, and to the stimulus *Oc* to remain practically unchanged The further shift to the position $A_2B_2C_2$ makes the response to the stimulus *Ob* subliminal, and diminishes the response to the stimulus *Oc*.

do not differ from one another by the circumstance that they bring into activity different groups of neurons, but above all because they produce different excitato-inhibitory configurations within the nervous system, in both a spatial and a temporal sense. It follows that, with the exception of so-called 'local reflexes', there are hardly any reflexes which are mutually independent of one another, and which, acting simultaneously, have no reciprocal relations. This truth becomes even more evident when we turn from the spinal animal to an animal with intact brain.

'It is a cardinal feature of the construction of the higher vertebrate nervous system', writes Sherrington, 'that longer indirect reflex-arcs, attached as extra circuits to the shorter direct ones, all pass through the brain. With those former intact the number of reflexes neutral one to another might be fewer. In presence of the arcs of the great *projicient receptors* and the *brain* there can be few receptive points in the body whose activities are totally indifferent one to another Correlation of the reflexes from points widely apart is the crowning contribution of the brain towards the nervous integration of the individual.' (Sherrington, *Integrative Action*, 1947, p. 149).

According to Sherrington we have to distinguish between *allied* reflexes, i.e. reflexes which act in the same manner on the given nerve cells, exciting or inhibiting them, and *antagonistic* reflexes, i.e. those which act on the same nerve cells in different ways. The interaction of allied reflexes results in their 'summation', while the interaction of antagonistic reflexes leads to 'interference' between them.

The principles of summation of two allied reflexes in relation to a single effector unit were discussed in §3. When a stimulus is applied to the receptors or the total centripetal nerve there is, of course, an excitation of many neurons, certain of them being excited more strongly, others more weakly. And so, if two allied reflexes act synchronously their general effect is more complex than in the case of excitation of a single cell, since it can depend on facilitation in certain cells and a lesser or greater occlusion in others But, generally speaking, the basic relations remain of course the same; two concurrent weak reflexes result chiefly in facilitation, while concurrent strong reflexes give rise to occlusion, this being the more complete, the stronger the stimuli applied.

However, it has to be remembered that alliance between reflexes exists not only when corresponding stimuli belong to the same 'receptive field' and provoke roughly the same excitation patterns in the nervous system. It can be detected also between reflexes exciting supraliminally quite different pools of neurons and possessing apparently quite different effects. For instance, stimulation of the forefoot in the spinal animal gives as primary

response a flexion of this foot, while the stimulation of the crossed hindfoot causes primarily flexion of that limb. And yet these two reflexes acting simultaneously reinforce each other and prove to be allied to a high degree The cause of this alliance is that each of these reflexes, exciting supraliminally a certain group of neurons, produces subliminal excitation in many other groups, *inter alia* in that group which is thrown into activity by the other reflex. This subliminal excitation does not manifest itself when the reflex acts separately, but betrays its existence when the reflex is combined with another allied reflex.

All this problem was thoroughly analysed by Sherrington in Chapter v of his *Integrative Action of the Nervous System.*

Let us pass now to the analysis of antagonism between reflexes. At one time, while studying the interaction between the flexion reflex and the scratch reflex, Sherrington expressed the view that the struggle between two antagonistic reflexes for 'the final common path' is always uncompromising, and that one or the other reflex dominates that path completely * Thus the antagonistic reflexes (according to his earlier view) can enter only into successive (and not simultaneous) combinations between each other, since only '*this* reflex or *that* reflex, but not the two together' can act at the same time. But later research by his school showed that this view was not correct, and that in the struggle between reflexes, one of which causes excitation and the other inhibition of a given group of neurons, compromises are reached which often resemble the 'algebraic summation' of two antagonistic processes. The precise mechanism of such compromises was discussed in the foregoing section.

The *total* victory of one of the competing reflexes occurs only when it possesses a considerable preponderance over the other. Thus interference between antagonistic reflexes appears in certain respects to be similar to the situation which arises when using a pair of scales. If the weight on one of the pans is greatly in excess of the other, it weighs down the pan completely, i.e. the other pan is completely neutralized, no matter what weight is in it. So in these cases one side has an uncompromising victory over the other. But within the range of a certain narrow

* Sherrington, *Integrative Action*, pp. 118 et seq.

ratio of relations between the two weights neither side achieves complete predominance, and the needle points to one or another position in its arc in dependence on fine differences in the weights put into the two pans. So in this case one side has only a relative victory, which can be greater or lesser. And just as in different scales this state of relative equilibrium lies within a broader or narrower arc, and can be achieved either more easily or with greater difficulty, so perhaps it varies for different kinds of reflexes. In certain instances it is very narrow, and then it is extraordinarily difficult to match up the strength of the two acting stimuli so as to ensure that neither of them predominates completely, and the struggle between them always appears to be uncompromising; but in other instances it is comparatively easy to reach this relative equilibrium between reflexes and, by correspondingly apportioning the strength of the two stimuli, the scale of victory can be shifted to one or the other side with comparative ease.

In the case of higher nervous activity this comparison with a pair of scales would seem to convey accurately the actual state of affairs, and it is a general rule for compromise to occur between two antagonistic reflexes if their relative strengths are appropriately matched

When speaking of alliance or antagonism between two reflexes we must remember that these concepts concern the action of the two reflexes only on a definite group of neurons in a certain period of time. Since, as we have said, every reflex represents its own peculiar (both temporally and spatially) and very complex configuration of excitations and inhibitions, it is hardly likely that with a normal activity of the organism any two reflexes would be exact copies of each other, or, on the contrary, exact negatives, i.e. that they would be absolutely allied or antagonistic with regard to *all* the effectors on which they work. Undoubtedly, in the great majority of cases, or perhaps always, two reflexes which are allied within the scope of one group of effectors can be antagonistic within another group, and may even be allied within a group of effectors at one moment and antagonistic at another. If we cannot cite many precise examples of this state of affairs, this is because research on reflexes has

hitherto been conducted chiefly so as to focus attention on one definite effector, or sometimes on two, but ignoring all others. None the less, even in his early work Sherrington draws attention to this state of affairs,* giving as example the interrelation between the scratch reflex and the ipsilateral flexion reflex.

We shall describe this fact in his own words. 'The scratch reflex, though at first sight it appears unilateral, is not strictly so Suppose the left shoulder stimulated, the left leg then scratches; but if the right leg is examined it is found to present slight steady extension with some abduction. ..Again the homonymous flexion-reflex of the hind leg (spinal dog) is only the main part of a larger complex reflex which is bilateral, and consists of flexion of the same side leg and extension of the crossed leg (the crossed extension-reflex). This being so, the mutual relation between the complete scratch-reflex, e.g. of left foot, and the complete noci-ceptive reflex of the same foot, is that the homonymous uncrossed parts of each reflex interfere and are related mutually as antagonistic reflexes, but the crossed parts of each reflex coalesce in excitation of the extensor neurons and inhibition of the flexor neurons of the right leg, and are related mutually as allied reflexes.'

When setting about the investigation of phenomena of higher nervous activity all these relations have to be remembered, since in the case of a normal animal with intact brain they are manifested especially clearly.

* Sherrington, C. S., *Integrative Action*, pp 145 et seq

CHAPTER V

Excitability and plasticity in the nervous system

1

Before we proceed to the main part of our discussion concerning the new approach to the physiology of higher nervous activity, it seems desirable to analyse more precisely than may have been done hitherto those functional properties of the cerebral cortex which distinguish this organ from the lower levels of the nervous axis, and which give its functions their specific character.

Let us begin by characterizing those properties, utilizing a comparatively simple cortical phenomenon, such as the extinction of an orientation reflex.

It is known that the higher animal reacts to external stimuli falling upon certain of its receptors (such as auditory or visual) by turning its head in the direction of the source of the stimulus, pricking up its ears, sometimes by a slight start involving all its body, etc. This is the so-called orientation reflex. Its strength can be measured either globally on the basis of the magnitude of the movements mentioned, or more accurately, but also more onerously, by investigating the extent to which this reflex inhibits an alimentary conditioned reflex evoked simultaneously.

If the stimulus causing the orientation reaction is repeated again and again, this reaction (measured either directly or indirectly) gradually diminishes and may even fade altogether. This process is called extinction of the orientation reflex.

There is no doubt whatever that the mechanism by which the extinction of the orientation reaction operates is *sui generis*, and that it must not be identified or even said to be analogous with other, apparently similar phenomena, such as the gradual disappearance of spinal reflexes through their too frequent repetition, the decrease of reaction in neuromuscular preparation kept in an unsuitable physiological condition, etc. For while in these latter cases the disappearance of the function is explained either by the lesion of the preparation, caused by fatigue or poisoning, or by a subnormal period of the excitability cycle

after the passage of nervous impulses, the extinction of the orientation reflex is not referable to any of these categories of phenomena. Many proofs of this may be adduced. First, we have clear experimental evidence that the orientation reflex is extinguished even when the intervals between the stimuli are very long (24 hours or longer) so that neither a functional exhaustion nor a subnormal period can account for this process. Secondly, there is evidence that the extinction of the orientation reflex occurs the more rapidly the better the functional condition of the nervous system and the higher the degree of its phylogenetic development. Thirdly, it is known that in animals whose cerebral cortex has been removed the orientation reflex extinguishes incomparably more slowly than in normal animals, and possibly is not extinguishable at all, being as stable as other unconditioned reflexes. Fourthly, and finally, as will be seen later there are phenomena profoundly analogous to the phenomenon of extinction of the orientation reflex, in which the reaction to a stimulus does not grow weaker on its repetition, but on the contrary grows stronger and stronger, which makes any analogy with fatigue or with a subnormal period quite inadmissible.

So we have to infer that in a normal animal the application of a stimulus evoking an orientation reaction leads to changes of a twofold kind in the nervous system. First, it gives rise to a cycle of acute changes, which consist in some transient excitato-inhibitory configuration arising in the nervous system, as the result of the given stimulation, and disappearing completely after a shorter or longer period. But, on the other hand the stimulus inaugurates certain permanent and irreversible changes in the centres, these having the effect that when, after some time, even a long time, the stimulus acts again, the excitato-inhibitory configuration elicited by it will now be somewhat different from that of the previous occasion The first property, by virtue of which the nerve cells *react* to the incoming impulses with a certain cycle of changes, we call *excitability*, and the changes arising in centres because of this property we shall call *changes due to excitability* The second property, by virtue of which certain permanent functional *transformations* arise in particular systems of neurons as the result of appropriate stimuli or their

combinations, we shall call *plasticity*, and the corresponding changes *plastic changes*. So among changes due to excitability we include such as a manifest or subliminal excitation of the nerve centres, inhibition of the centres caused by, for instance, an antagonistic reflex, changes of their excitability caused by the excitability cycle, etc.; but among plastic changes are the extinction of the orientation reflex, the formation of active and inhibitory conditioned reflexes, habit formation, etc.

As is well known, excitability is a universal feature of the nervous system, whereas plasticity is a characteristic of only its highest divisions, and, in the higher vertebrates, especially of the cerebral cortex.

It is remarkable how little attention physiologists have devoted to the problem of plasticity in the sense just given. While investigations concerning the excitability properties of the cerebral cortex are numerous and manifold, and by various methods a great mass of facts has been gathered in this field, the study of plasticity in the functioning of this organ is as yet but little advanced. Even Pavlov, who, having introduced the conditioned reflex to physiology, was particularly concerned with a phenomenon which is specifically due to plasticity, who fully appreciated the revolution he had brought about, and gave this property of the cerebral cortex the name 'linkage' ('Zamykatielnost')—even he did not devote adequate attention to the problem of this 'linkage', and most of the investigations of his school were devoted to the excitability properties of the cerebral cortex, studied with the method of ready-made conditioned reflexes.

We cannot now go into a detailed discussion of the reasons for this state of affairs. Perhaps it is partly due to intellectual inertia, which impels the physiologists who study the activity of the cerebral cortex to follow beaten paths and seek in the functioning of this organ the same problems that were known from the study of the lower parts of the nervous system. Furthermore, it must be realized that, unlike excitability, the plasticity of the cortical activity has so far not yielded to *direct* investigation. The mechanisms of plasticity are to be found only in a normal animal, whereas after it has been anaesthetized and its

skull has been opened they vanish completely, and so the experimenter who is tempted to make direct investigations into the activity of the cerebral cortex finds that under his hands it becomes only an excitable organ.*

2

As we have said, the extinction of the orientation reflex (and of certain other unconditioned reflexes) is not the only type of plastic phenomena. There are other types, some of which have been examined far more thoroughly. Thus, if two stimuli are applied always in overlapping sequence a plastic change occurs in the nervous system, in that the 'leading' stimulus begins to evoke the same reaction as the 'aftercoming' stimulus (conditioning). If the conditioned stimulus is given separately without the reinforcing stimulus, a plastic change occurs, consisting in the disappearance of the conditioned reflex (internal inhibition). Further, there is a more complex kind of plasticity, on which the habit formation is based, to which we have given the term 'conditioned reflexes of the second type'; this kind will be discussed in one of the final chapters Undoubtedly there are other kinds of plasticity also, which so far physiology has not concerned itself with, but which are well known to psychologists. All of them, without regard to whether they result in the establishment of a new reaction to the stimulus, or, on the contrary, in a subsiding of the former reaction, or both one and the other, possess certain general common properties which make it possible to treat them as one great class of phenomena, probably of identical or similar nature.

Here we shall specify some of these properties·

(1) If the application of a certain combination of stimuli (here we include an individual stimulus in the term combination) tends to give rise to a definite plastic change, the repetition of

* In this direction a beginning has been made by the work of those experimenters who are adapting the method of direct stimulation of the cerebral cortex to investigations on conditioned reflexes (Loucks, R B., *J Psychol* 1935, vol. I, p 5, *J Comp Psychol* 1938, vol XXV, p. 315; Konorski, J and Lubinska, L , *Acta Biol. Exper* 1939, vol XIII, p. 143, and others) So far, however, it is difficult to say what this line of research will yield

this combination leads to 'cumulation', i.e. an increase in this change. The cumulation of a plastic change depends on the number of repetitions of the combination, and on the intervals between the separate trials.* The smaller the intervals, the swifter occurs the cumulation of the plastic change However, *too* frequent trials are detrimental to the formation of the plastic change, because of the proneness to fatigue of the nervous elements involved.

Examples. (*a*) The application of a combination, consisting of an indifferent stimulus and an unconditioned stimulus in overlapping sequence, leads to the formation of a conditioned reflex, which increases *pari passu* with the repetition of the combination The more frequently the combination is given, the more rapid is the formation of the reflex, i.e. the fewer the trials required, however, too frequent trials do not lead to an acceleration of the establishment of the reflex, but rather they delay its formation.

(*b*) The repetitive application of the same conditioned stimulus without reinforcement leads to extinction of the reflex, and the extinction deepens *pari passu* with the repetition of the stimulus. The shorter the intervals between the trials (within certain limits) the fewer the trials required to achieve the complete extinction of the reflex. By way of illustration we give extracts from Babkin's experiments.†

The conditioned stimulus consists in showing a dog meat powder for 1 minute. This stimulus is not reinforced.

If the stimulus is given every 16 minutes it is not completely extinguished after seven trials.

If it is given every 8 minutes it is completely extinguished after six trials.

If it is given every 4 minutes it is completely extinguished after four trials.

If it is given every 2 minutes it is completely extinguished after five or six trials.

* By 'trial' we mean each application of the stimulus or combination of stimuli, which leads to the formation or maintenance of some plastic change.

† Babkin, B P , Diss , Petersburg, 1904 (Pavlov, *Conditioned Reflexes*, p 53)

Thus, in these experiments the optimum interval of time for the extinction of the conditioned reflex was 4 minutes.

(2) If the combination of stimuli which is the cause of the plastic change is not applied, this change suffers *regression*, which is the stronger, the longer the interval since the last application of the combination. If the interval is long enough there may even be apparently complete regression. That it is only apparent is shown by the fact that a reapplication of the combination leads to the formation of the plastic change more rapidly than on the first occasion

Examples. (*a*) A combination is given consisting of an indifferent and an unconditioned stimulus. After a number of trials a conditioned reflex of a certain magnitude is formed. A suspension in application of the combination causes a weakening of the conditioned reflex; after a sufficiently long interval the reflex can disappear altogether. But if we try to form it anew, it is established more rapidly than on the first occasion.

(*b*) A stimulus evoking an orientation reaction is given at intervals of several minutes. After a number of applications the orientation reflex extinguishes completely. If after an hour's interval the same stimulus is again applied, it may be found that the orientation reflex has recovered to a greater or lesser extent. After an interval of many hours the recovery may be complete If now the same stimulus is applied at the original intervals, it will be found that the reaction to it subsides much more rapidly than before.

(3) The longer the period over which the combination of stimuli causing a plastic change is repeated, the more *stable* the change becomes, i.e. there is less regression if the application of the combination is suspended.

Example. While a recently established conditioned reflex undergoes regression very swiftly, almost from day to day, a conditioned reflex trained over a long period hardly suffers regression at all, i.e even a break of many months in the application of the combination does not cause any diminution in its effect.

The differences in stabilization of plastic changes arising from the degree of training are of great importance in work on con-

ditioned reflexes, and provide a straightforward explanation for facts which the Pavlov school have never interpreted from this viewpoint. Take, for example, the spontaneous recovery of an extinguished conditioned reflex after a suspension in application of the conditioned stimulus. From the viewpoint of the laws discussed above, this fact is very easily explained. The positive-conditioned reflex constitutes a plastic change firmly established, and therefore even longer intervals between the trials are of no significance for its maintenance, on the other hand, the extinction of this reflex is a fresh plastic change, which undergoes regression very rapidly. Consequently, after a couple of hours' interval in the application of the conditioned stimulus the extinction undergoes apparently complete regression,* but in practice the conditioned reflex suffers no regression whatever. As the result a complete recovery of the conditioned reflex occurs. Obviously, if the conditioned stimulus is regularly given day after day without reinforcement, then the new plastic change, i.e. the extinction, grows more and more fixed and becomes just as stable and insensitive to a suspension in the application of the stimulus as was the active conditioned reflex originally.

3

Accepting plasticity as an independent property of the functioning of higher nervous activity, we must also admit that it can be displayed in various degrees in different species of animals, in different individuals of the same species, and even in the same individual in different states, or at various periods of its life. In fact, a considerable body of experimental evidence indicates that animals at the lower stages of phylogenetic development form both active- and inhibitory-conditioned reflexes, and habits also, more slowly than higher animals, that there are great differences in the rate of formation of plastic changes among various individuals of the same species, that the plasticity of the nervous system grows feebler as the result of illnesses,

* Apparently, because if we tried immediately to extinguish this same reflex a second time, the extinction would occur much more rapidly than on the first occasion

through organic lesions of the nervous tissue, in states of fatigue, in old age, etc.

Just as the physiology of higher nervous activity has hardly paid any attention to the general problems of plasticity, so it has completely ignored the questions concerned with the rate of formation of plastic changes, or the rate of their regression. Thus, whereas in psychology experimental research into memory is regarded as very important, and constitutes one of the best explored departments of this science, physiology so far has had little to say on the subject. One gets the impression that physiologists have even avoided raising clear issues in this field. Pavlov, for instance, when analysing the greater or lesser rate of formation of positive or inhibitory conditioned reflexes, attributes it to the excitability of the animal's nervous system, to the 'strength' of the processes of excitation or inhibition, to their 'lability' (see below). But it never occurred to him that capacity to form plastic changes is a property *sui generis*, which can be correlated with other characteristics of the nervous system, but must not be identified with them

How great was the misunderstanding of problems of plasticity in the Pavlov school can be seen from the paper written by one of Pavlov's pupils, E. Asratian, 'On the physiological lability of the higher central levels'.* In this paper the author compares the rate with which conditioned reflexes are established, extinguished, delayed, differentiated, etc., in two dogs of approximately the same age and identical 'strength' of their nervous processes (in the Pavlov school sense), and shows that the formation of both positive and inhibitory conditioned reflexes in one of the two dogs proceeded at a much faster rate than in the other. Reading the reports of these experiments, one can have no doubt whatever that this was a case of a difference in the plastic properties of the two animals; yet the author argues that the different rate of formation of reflexes depends on ..the lability of their nervous systems. By 'lability' (a term used by Wedensky) he means the property corresponding to Lapicque's 'excitability' and measurable by chronaxie. The author himself states that 'right at the beginning

* Asratian, E., *Uchonie Zapiski L. G. U.* 1939, no. 41, p. 23

a question of first-rate importance has to be asked. to what extent are we entitled to accept the comparative rate of reaction development by the method of conditioned reflexes as lability in Wedensky's sense?' Without providing any convincing evidence Asratian none the less regards these two phenomena as identical, and by so doing attempts to squeeze into the framework of the physiology of the lower nervous activity that very property of the cerebral cortex which is almost exclusively specific to it.

So the problem of plasticity of the nervous system is almost an unexplored region for physiology, and research in this direction should yield valuable results. Whether or not the lower species of animals really show a lesser plasticity than the higher (as is universally accepted), whether or not the relative rate of formation of plastic changes is identical for various kinds of plasticity, what influence humoral factors, the autonomic system, etc., have on plasticity, what correlation exists between plasticity and other properties of the nervous system—all these are problems which can and should be investigated with the aid of physiological methods, and their investigation from this aspect will undoubtedly be of great benefit.

4

Remembering the poor development of physiological investigation into plasticity, one cannot be surprised that in the present state of knowledge very little can be said about the intimate nature of this phenomenon. If none the less we raise the matter here it is because a general view of the mechanism of plasticity is an indispensable link in the construction of a theory of the functioning of the cerebral cortex. In fact, as the present work is devoted to the plastic phenomena arising in the cortex, it is impossible to pass over the problems of their general mechanism.

For the purpose of considering the mechanism of plastic changes we shall concentrate attention on the formation of conditioned reflexes, postponing the problem of their inhibition and the inhibition of the orientation reaction to one of our later chapters.

There would seem to be little doubt that in the present state

of physiological knowledge the most simple and natural explanation of the elaboration of conditioned reflexes is the assumption that between the centre of the conditioned stimulus and the centre of the unconditioned stimulus previously non-existent functional connexions are formed. This is suggested both by the existence of spatially separate 'centres' for different stimuli (i.e. of assemblies of cells, scattered over the nervous system, which on the application of the given stimulus are regularly excited), and by the fact that there is a great similarity in physiological properties between the well-established conditioned reflexes and ordinary reflexes, which implies a similar structure of their reflex arcs The nature of these connexions is a matter for discussion. Obviously, it is hardly to be believed that, as the result of repetitive application of one or another combination of stimuli, new, previously non-existent, *nerve paths* could arise in the cerebral cortex. There is clear morphological evidence against this assumption, and, moreover, the very fact of the sometimes extraordinarily rapid formation of the conditioned reflex (even after one association of adequate stimuli) points to its extreme improbability. Thus we have to assume that the formation of new intercentral connexions must be regarded as *a synaptic process*, i.e. only such groups of neurons as are predestined to this because of corresponding anatomical paths can be brought into a functional relationship. In other words, we have to assume that before a conditioned reflex is established, there exist *potential* interneuronic connexions, directed from the centre of the stimulus to be conditioned to the centre of the reinforcing stimulus, and that as the result of repetitive associations of these stimuli these potential connexions are transformed into *actual* connexions. The neurons or centres linked together by such potential connexions we shall call *coupled*, and we shall distinguish the *emitting* neuron or centre, which sends impulses to the other centre, and the *receiving* neuron or centre, to which these impulses are addressed.*

* Ariens Kappers (*Brain*, 1921, vol. XLIV, p 125), discussing the problem of selectivity in the formation of new connexions as a result of simultaneous excitation of two centres, called such centres 'stimulatively correlated'. We think, however, that the term 'coupled' we propose is more suitable and more expressive.

The problem arises: What is the intimate nature of the transformation of potential into actual connexions? Two main lines of approach to the solution of this problem can be distinguished morphological and functional.

The functional interpretation of plastic changes has been generally more popular among physiologists than the morphological interpretation, and these changes were most frequently related to the phenomenon of facilitation. This view, however, is rather difficult to maintain, since facilitation represents, strictly speaking, nothing else than one of the phases of the excitability cycle, and as such is fully reversible, in contradistinction to the practical irreversibility and permanence of plastic changes. In recent years more serious attempts have been made to explain plastic changes in functional terms by the assumption that the formation of new functional connexions is based on bringing into activity previously inactive closed self-re-exciting chains of neurons in the highest divisions of the nervous system * According to this hypothesis simultaneous application of two stimuli (the one to be conditioned, and the other unconditioned) initiates activity in certain closed chains of neurons, which from that moment continue to function unceasingly. Their functioning has the result that when the conditioned stimulus is now applied alone it evokes a response which previously it did not produce. The cessation of this steady activity corresponds to the phenomenon of forgetting. Models of interneuronic connexions are constructed to account for the processes of conditioning, extinction and so on.

It seems to us that this theory presents difficulties (indicated already by Householder and Landahl) which cannot be overcome. In fact, if we hold that the whole body of interneural connexions acquired by the organism during its individual life is due to the incessant activity of self-re-exciting chains of neurons, once thrown into activity, it is incomprehensible how they can be preserved after such states of complete inactivity of the brain

* Vide Young, J. Z., in *Evolution, Essays on Aspects of Evolutionary Biology*, Oxford, 1938, p. 179, Rashevsky, N , *Mathematical Biophysics*, Chicago, 1938; Householder, A. and Landahl, M , *Mathematical Biophysics of the Central Nervous System*, Bloomington, 1945

as are produced by a very profound narcosis, a cerebral ischemia, and so on. We know that in these states very simple and primitive functions of the nervous system (such as two-neuron reflexes) are completely abolished and precluded, so how is it possible that highly complex and delicate activities could continue to be sustained? And yet it is well known that the whole body of 'engrams' is as a rule completely preserved after such states, and that the various forms of partial amnesias are described and investigated rather as exceptional phenomena, they being in most cases fully reversible.

In our opinion the objection just raised not only renders inadmissible this particular conception, but also must be taken into account in discussing any other conception attempting to explain plastic changes in functional and not morphological terms.

On the other hand, reasonable arguments can be put forward in favour of a morphological conception of plasticity, according to which plastic changes would be related to the formation and multiplication of new synaptic junctions between the axon terminals of one nerve cell and the soma (i.e. the body and the dendrites) of the other.*

So the significance of training in the establishment of new connexions, the great dependence of the plastic properties of a nervous system upon the age of the organism, the fact that in some pathological cases (such as Korsakoff psychosis, pellagra, etc.) the formation of new plastic changes is greatly impaired while the formerly elaborated changes are preserved, all these facts are in agreement with the morphological hypothesis of

* As is well known, such a view was put forward by Ramon y Cajal, S., *Textura del sistema nervioso del hombre y de los vertebrados*, Madrid, 1904, Ariens Kappers, C U., *J. Comp. Neurol.* 1917, vol XXVII, p. 261; *Brain*, 1921, vol XLIV, p. 125; Child, C. M., *The Origin and Development of the Nervous System*, Chicago, 1921; Coghill, C E., *Anatomy and the Problem of Behaviour*, Cambridge, 1929. From the last of these authors we give the following noteworthy quotation (pp 84–5): 'Growth of the terminals of axons and dendrites through microscopic dimensions is sufficient to have a profound effect in behaviour. This we have demonstrated in a vertebrate of such primitive form as ambylastoma, which, by the growth of terminals of nerve cells over a distance of less than one one-hundredth of a millimetre, transforms itself from an animal that must live helpless where chance places it into one that can explore its environment in response to impulses from within or stimulation from without'

plasticity. The phenomenon of the regression of plastic changes when the appropriate training is suspended (which fact can be considered as homologous to the general phenomenon of 'atrophy from disuse') also fits in very well with this hypothesis, as does the fact that the regression chiefly affects fresh plastic changes leaving the old ones rather undisturbed.*

Taking all the foregoing into account, we are rather disposed to accept the morphological hypothesis of plasticity as the basis of our further discussion, the more so because, as will be seen later, it is suitable for the systematization and explanation of the body of facts provided by the physiology of higher nervous activity.

Thus, according to the theory we have adopted, we shall use the expressions 'formation and multiplication of synaptic connexions' to denote the processes underlying the elaboration of conditioned reflexes (or other plastic changes), and 'fading or atrophy of synaptic connexions' to denote the processes underlying the regression of plastic changes when the appropriate combination of stimuli is withheld It must be stressed, however, that when using these expressions we are not necessarily bound to assign them strict and concrete morphological meaning, and we think that they may retain their usefulness even if some other theory of plastic changes proves to be adequate.

5

We have said that actual connexions between two neurons can arise only when they are coupled, i.e when potential connexions formed owing to the hereditary patterns of development already

* Compare Ramon y Cajal's 'law of atrophy through disuse' of the newly formed axon when 'it has not been able to establish congruent anatomico-physiological relations with the terminal structures' (Ramon y Cajal, S., *Degeneration and Regeneration of the Nervous System*, London, 1928, p. 367). In this connexion it is also worth drawing attention to Young's 'Principle of double dependence' (*The Lancet*, 1946, p 109). This author indicates that the normal functional condition of nerves is ensured when they receive some stimulation from above and exercise some stimulating effect below, and that, if one of these factors fails to act, atrophy of the tissue occurs. I think that the analogy between this principle and the actual condition of the maintenance of a conditioned reflex is so manifest that it must be considered as something more than a merely superficial coincidence.

exist between them. The question arises whether the cerebral cortex is so constructed that *any* of its centres can form conditioned connexions with any other centre, or whether potential paths exist only between definite centres, and the existence of these paths predetermines the bounds within which further development, due to the individual experiences of organisms, can proceed.

No satisfactory answer can be given to this question. It is well known that conditioned reflexes reinforced by such unconditioned stimuli as the presentation of food to the animal, the stimulation of the paw with an electric shock, etc., can be formed to any extero-, intero- and proprioceptive stimuli, and that perhaps there is no stimulation of an animal's receptors which could not be conditioned. Moreover, the experiments of Narbutovich and Podkopayev* and other, similar observations (vide Chapter XI, § 3) indicate that in dogs 'indifferent' stimuli, visual, auditory, tactile, etc., can also be mutually conditioned. Similarly, the many years of research work of the Bykov school† shows that any functions of the organism controlled by the central nervous system can serve as a basis for the formation of conditioned reflexes All this evidence indicates that the possibilities provided in the field of conditioning by the brain of such an animal as the dog are really enormous, and on the basis of these data we are disposed to believe that in fact there are no reciprocally unrelated areas whatever in the cerebral cortex.

But, on the other hand, it must be remembered that conditioned reflexes of the Pavlov type do not exhaust all the acquired activity of the higher animals, and that there are forms of cortical activity which are peculiar to species with a higher cortical structure and are not found in animals on the lower levels of phylogenetic development It is difficult to say whether

* Narbutovich, I. O and Podkopayev, N A , *Trudy Lab Pavlova*, 1936, vol. VI, no 2, p. 5; these authors applied concurrently stimuli evoking an orientation reaction (light and tone, hissing and the sight of a rotating object, etc) and found that after a number of trials the application of one of these stimuli caused a reaction in the direction of the source of another.

† Cf Bykov, K. M., *Kora golovnovo mozga i wnutrennie organy* (*Cerebral Cortex and Visceral Functions*). Moscow, 1944.

such differences are due to the existence or absence of corresponding nerve paths, or a greater or lesser development of particular centres.

It is just as impossible to answer another question, namely, whether various centres of the cerebral cortex are always coupled with one another in the same manner, or whether in certain cases the potential connexions are more direct, whereas in other cases they are intercalated by more or less numerous intermediary links. The fact that in certain cases conditioned reflexes are formed extraordinarily slowly and obstinately, whereas in other cases they are quite established after one or two trials, would seem to suggest the second alternative But as the differences in the rate of formation of conditioned reflexes can often be explained without appealing to a greater or lesser complexity of nerve paths between centres, this question also must remain undecided.

However, the problems to which this monograph is devoted do not require answers to the questions just asked. So we content ourselves with stating that potential connexions do exist between various centres of the cerebral cortex, and that they provide an anatomical basis for conditioning. Moreover, for the sake of simplicity in our further consideration, we assume that the coupling between such centres is complete, i.e that *each* neuron of the emitting centre is potentially connected with *all* the neurons of the receiving centre. Indeed, we see no adequate reason to assume any arbitrary hypothesis of only partial connexions between the coupled centres, and besides, such an assumption would greatly complicate our further discussion. Whether *any* cortical centres are mutually linked by potential connexions, and whether the connexions between various centres possess various degrees of complication (in the sense of different numbers of intercalated links) are problems which for the time being remain unsolved.

CHAPTER VI

The conditioned reflex

1

As we have said in the previous chapter, the simplest and best known type of plasticity is the Pavlovian conditioned reflex. Its formation consists in the establishment of new functional connexions between two concurrently excited groups of nerve cells, one representing the centre of the conditioned stimulus, the other being the centre of the reinforcing unconditioned stimulus. For the sake of convenience we shall call the first of these centres simply the 'conditioned centre', and the second the 'unconditioned centre'.

Here we shall not prejudge the question either of the structure possessed by these centres or of their localization Therefore we shall not consider the problem whether, for a conditioned bond to arise between them, both the conditioned and the unconditioned centre must be situated in the cerebral cortex (as Pavlov was inclined to think), or whether conditioned 'cortico-subcortical' or 'subcortico-subcortical' connexions can also exist. We point out, however, that, in our opinion, in a normal mammal a dominating role in conditioning must unquestionably be attributed to the cerebral cortex.

In stating that the conditioned reflex is due to the formation of new intercentral connexions (in the sense determined in the preceding chapter), we have no intention of implying that these connexions are direct and simple. On the contrary, we have many reasons for thinking that the conditioned reflex is an extremely complex nervous phenomenon, and we must remember that its schematic simplification, although in many cases useful, is really only a fiction. Let us specify the main factors which condition this complexity. First, the so-called conditioned stimulus, such as is employed in experiments, never acts in isolation, but always together with a compound of other stimuli, constituting the environment of the experiment. That these share in the conditioned reflex is shown by the fact that the

same conditioned stimulus used in another environment is more or less, or may be even entirely, deprived of its conditioned action. As a matter of fact, the conditioned stimulus is always a certain complex situation, acting on a number of receptors, a single element of which is artificially isolated out and subjected to analysis Secondly, even if we take into consideration only the conditioned stimulus in this more strict sense, its action on the cerebral cortex is not restricted to the excitation of a single cortical *point* (as Pavlov liked to say) or to a restricted group of cells. On the contrary, we have no doubt that the formation we briefly term a conditioned centre is extremely complex and multi-storied, and that cell groups of different cortical fields belong to it. More exact evidence indicating this will be given in one of the later chapters The same applies to the unconditioned centre. It hardly needs to be mentioned that an excitation of this centre causes not only the response on which our attention in experiments is chiefly concentrated, but also many other responses, within the range of almost all the animal's effectors, so the treatment of an 'unconditioned centre' as a spatially restricted and defined morphological group of cells would be quite obviously absurd Finally, as we have already said, we assume that in the majority of conditioned reflexes the path from the conditioned to the unconditioned centre leads through a number of 'intermediary stations', i.e. internuncial centres, each of which must also possess a complex structure.

It follows from all this that the structure of the conditioned reflex is far from simple, and we are still a long way from knowing its precise nature. And so, not making any attempt to follow up this question, we confine ourselves to a certain simplification of the conditioned reflex, concentrating attention solely on its chief links, namely the conditioned stimulus in the stricter meaning of the words, the cortical centre of this stimulus, the centre of the unconditioned stimulus (each of these centres being treated globally, without going into their structure) and the definite conditioned effect. We deliberately ignore all the other component parts of the conditioned reflex, referring to them only when we need them in order to explain experimental facts.

2

In this chapter we intend to present the fundamental properties of the conditioned reflex, and to attempt on their basis to elucidate the mechanism of the formation and course of this reflex in more detail than in our previous chapter.

We begin with those properties of the conditioned reflexes that are attached to their formation and training, i e. their plastic properties.

If we form a conditioned reflex, reinforcing the 'indifferent' stimulus by an unconditioned stimulus of constant strength, the conditioned reflex gradually grows, until finally it reaches a certain magnitude which is its maximum. We shall call this magnitude the *limit value* of the given conditioned reflex. The limit value is not entirely constant, it may fluctuate to a smaller or greater degree, in dependence on the temporary state of excitability of the nerve centres coming into play, but it does not depend on the number of applied associations of the stimuli, as is the case in the initial phases of conditioning.

Given a constant strength of the reinforcing stimulus, the limit value of a conditioned reflex is the greater, the greater the strength of the applied conditioned stimulus (the law of strength of conditioned stimuli, vide Chapter II, § 5). If the strength of the conditioned stimulus is increased, the conditioned reflex grows, as we recall, only to certain limits. Following Pavlov, we shall call these limits the *top*, but not the top of capability of cortical cells, as he called it, but simply the top of the conditioned reflexes. So the top of conditioned reflexes is the highest achievable value of these reflexes with the given excitability of the nerve centres and the given reinforcement. This value may equal the magnitude of the reinforcing unconditioned reflex.

The magnitude of the established conditioned reflex depends also on the strength of the reinforcing unconditioned stimulus (concretely, on the tastiness or abundance of the presented portion of food, the concentration of the acid poured into the mouth, the strength of the electric shock, etc.). The stronger the unconditioned stimulus, the greater, *ceteris paribus,* are the conditioned reflexes formed on its base.

The fact that a conditioned reflex has achieved its limit value by no means signifies that on its further repetition it will not be subject to further changes. We have much evidence that the further repetition of a reinforced conditioned reflex leads to its further stabilization. On the one hand, this stabilization causes the old conditioned reflex to fade much more slowly when the stimulus is not applied, than does a fresh reflex (vide p 83), and on the other hand (as many fortuitous observations have shown) when a conditioned reflex is firmly established it yields far less both to external inhibition and to experimental extinction than when it is fresh.

The foregoing is a brief outline of the basic facts observed in the formation of a conditioned reflex. We shall try to explain them, taking into consideration those arguments and data we have discussed in the two preceding chapters.

Before setting about this task, we must clarify more exactly what is meant by the concept of the strength of a conditioned or unconditioned stimulus. In harmony with the evidence of the physiology of lower nervous activity, we assume that a stimulus leading to a stronger reaction involves excitation of a larger number of cell elements in the given centre, i e a larger *neuron field,* while a weaker stimulus involves excitation of a smaller neuron field. Of course there is no doubt that the greater or lesser effect of a stimulus also depends on the intensity of excitation of each individual neuron, in other words, on the density of impulses reaching it. But, in order not to complicate our further discussion, this factor will be ignored.

On the basis of the foregoing assumption, it is easy enough to explain the fact of the relationship between the size of the conditioned reflex and the strength of the reinforcing unconditioned stimulus. In fact, as follows from the evidence of the preceding chapter, with a concurrent activation of two coupled centres each element in the conditioned centre becomes actively connected with all the elements of the unconditioned centre. Therefore, the stronger the unconditioned stimulus, i.e. the larger the cellular field excited by it, the greater the magnitude of the conditioned reflexes formed with its aid.

But explanation of the relationship between the magnitude

of the conditioned reflex and the strength of the *conditioned* stimulus, given a constant reinforcing stimulus, calls for more extensive consideration. For the neuron field of the unconditioned centre excited by the conditioned stimulus is in this case always the same, irrespective of the strength of this stimulus, so how is it that the reflexes to different conditioned stimuli are of different magnitudes?

To find the answer to this question it is necessary to consider more closely the rate of the formation of synaptic connexions when a conditioned and an unconditioned stimulus are repeatedly associated.

Two different possibilities seem to be reasonable and deserve discussion. (1) We can assume that the increment of synaptic contacts between two coupled neurons is roughly constant for each trial, depending only on the functional condition existing in the given centres at the moment of excitation. In this case the quantity of synapses formed between the neurons would be a linear function of the number of their simultaneous excitations (fig. 5*a*). (2) The increment of synaptic contacts between two coupled neurons on each concurrent excitation depends on the quantity of contacts already formed, diminishing with the increase of this quantity in such a manner that the total quantity of synapses between them approaches a definite limit (fig. 5*b*).

It is easy enough to show that acceptance of the first of these assumptions involves the conclusion that conditioned reflexes to any conditioned stimuli (from the weakest to the strongest) should possess an identical limit value, equal to their top value

Indeed, let us denote the increment of the synaptic connexions between two coupled neurons on each trial by k and the number of neurons comprised in the conditioned centre by n. Then the total quantity of synaptic connexions formed around a single neuron of the unconditioned centre after x trials will be $y = nkx$. Assume that Y denotes the quantity of synaptic connexions which ensures the maximal response of the given effector-unit. This value will be reached for any n provided that a sufficiently great number of trials is given. This means that with any strength of the conditioned stimulus the conditioned reflex must ultimately attain its top value. The difference between weak and

strong conditioned stimuli would consist solely in the different rate of attainment of this maximal effect; for the larger the cellular field representing the conditioned centre, the more new synaptic connexions are supplied to the individual neurons of the unconditioned centre at each trial.

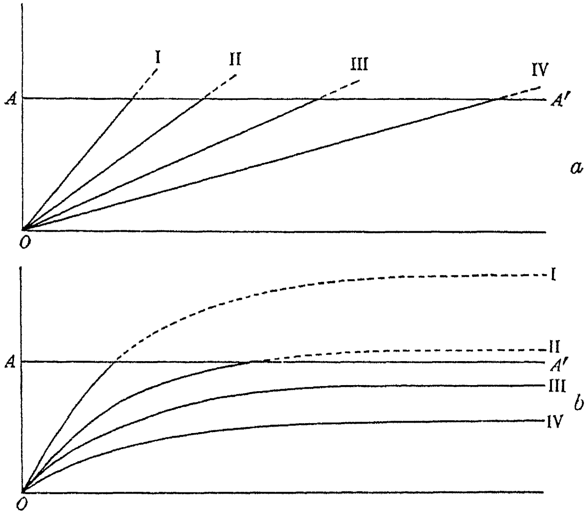


Fig. 5. Two hypotheses of the development of conditioned connexions. Abscissae: successive trials. Ordinates: quantity of connexions formed between the conditioned and the unconditioned centre. *OA*, the quantity of connexions necessary to reach the top value of conditioned reflex (*a*) The development of synaptic connexions on the assumption that the rate of their formation is constant The conditioned reflex reaches top value with *any* strength of conditioned stimulus, which contradicts the experimental data. (*b*) The development of synaptic connexions on the assumption that the rate of their formation decreases, tending to *O* In this case the limit value of the conditioned reflex depends, within limits, on the strength of conditioned stimulus *I*, *II*, *III*, *IV*, curves corresponding to different strength of conditioned stimulus

Because, as is well known, the ultimate value of a conditioned reflex to a given stimulus does depend on the strength of the conditioned stimulus, and because this dependence remains even after the two stimuli have been associated hundreds of times, we infer that the increase in synaptic connexions cannot occur linearly.

Let us consider now the other assumption, according to which only a limited number of synaptic contacts, *a*, can be formed between individual neurons of two coupled neuron fields. Then the ultimate quantity of synaptic contacts formed around a single neuron of the unconditioned centre will be *na*. If *na* is greater than *Y*, then the conditioned reflex will reach the top value after a sufficient number of trials; but if *na* is less than *Y*, then the ultimate effect of the given conditioned reflex will be less than the top effect and will depend on the number *n* of elements of the conditioned centre, i.e. on the strength of the conditioned stimulus. The relationship between the strength of the conditioned stimulus and the magnitude of the conditioned response would thus be fully confirmed

So we are led to the conclusion that the increase of synaptic contacts between two coupled neurons (or centres) repeatedly excited in association is limited. Although the actual shape of the curve representing this increase is still unknown, it can be reasonably assumed that the increasingly slower rate of formation of new synaptic contacts depends on the increasing number of contacts already established.

To make this last point more clear let us take into consideration the following explanation of the phenomena above discussed, which explanation can at least serve as the model of events, if one is not disposed to accept it as their true mechanism.

Assume that the concurrent excitation of two coupled neurons is accompanied by the emergence of electrical gradients between the axon terminals of the emitting neuron and the soma of the receiving neuron, and that these gradients are responsible for the formation of synaptic contacts.* If it is accepted that synaptic contacts already established between the axon terminals and the soma lessen the electrical resistance between them, we have to conclude that, as the synaptic connexions increase in number, the potential gradient diminishes. From this two important consequences follow. The first is that the formation of

* A similar view was extensively developed by Ariens Kappers and by Child (op cit) The experimental evidence of the influence of electrical gradients upon the nerve growth *in vitro* was recently produced by Marsh, G. and Beams, H. W., *J. Cell Comp Physiol*, 1946, vol xxvii, p. 138.

7-2

new synapses between two coupled neurons should proceed more and more slowly with the establishment of more and more synapses between them. Secondly, the synaptic contacts between the axon of one nerve cell and the soma of another should be dispersed over the soma of the cell, and not concentrated, since the formation of a new synapse close to an already existing synapse of the same axon is rendered impossible by the small potential gradient prevailing in this zone Consequently, on small zones of the neuron surfaces assemble only axon terminals belonging to *different* axons.

The latter of these statements was put forward by Lorente de Nó on the basis of histological research. The first we have just inferred from the analysis of facts obtained in the study of conditioned reflexes.

Now if we accept Lorente de Nó's conception, according to which the postsynaptic impulse is originated in the nerve cell when a sufficient quantity of presynaptic impulses is collected simultaneously on a discrete zone of its surface, then the relationship between the strength of the conditioned stimulus and the magnitude of the conditioned response will be made particularly clear. For the larger the neuron field excited by a conditioned stimulus, the more impulses are collected on every discrete zone of the neurons of the unconditioned centre and the better will be the conditions for their activation.

The maximal density of synaptic contacts connecting two coupled neurons we shall call their *saturation point.*

The foregoing considerations seem to make clear the fact of the invariability (in the sense of untrainability) of certain inborn reflexes, in which interneuronic connexions have reached the saturation point because of the hereditary scheme of development of the organism, the fact of the 'maturation' of other inborn reflexes, in which the interneuronic connexions have not reached the saturation point, and the formation of 'purely' acquired reflexes, in which all connexions have been potential.

Now only the last of the properties of the conditioned reflex, namely, its stabilization by the repetition of associations of the conditioned and unconditioned stimuli, remains to be explained. Unfortunately, it is difficult to analyse this process, because of

the small amount of existing factual material, so we can only put forward certain suppositions which need to be verified by future special experiments.

It seems that we have to distinguish two completely different aspects of the stabilization of conditioned reflexes. One is the growing resistance of the conditioned reflex to a break in its repetition. Although this aspect has not been submitted to systematic quantitative investigation, it is perfectly well known from everyday laboratory practice. The other, much more obscure, aspect is the increasing insusceptibility of the reflex to destructive influences, such as external and internal inhibition.

It seems that the first of these two facts—the growing permanence of the reflex in the sense of its not yielding to regression—should be attributed simply to a growing stabilization of the synaptic contacts. In fact, once we have accepted that synapses are formed through conditioning, and that they are subject to atrophy when the conditioned reflex is not trained, we see no difficulty in accepting that the training of a reflex leads to its increasing stability.

As for the increasing insusceptibility of the conditioned reflex to the inhibitory influence of extraneous agents, and its increasing resistance to internal inhibition, we assume that the mechanism of this phenomenon is quite different As will be shown in following chapters, in such cases the greater or lesser conditioned response depends on the result of a struggle between the impulses exciting and inhibiting the nerve cell. The greater the number of synaptic connexions through which excitatory impulses are transmitted to the neuron, the weaker will be the influence exerted on it by the inhibitory agent. As follows from the foregoing, the fact that a conditioned reflex has achieved its maximal value does not mean that the increase in synaptic connexions between concurrently activated centres is brought to an end, for, given a sufficiently strong conditioned stimulus, the number of these connexions may prove adequate to evoke the maximal response even at earlier stages of conditioning. So a further increase in synaptic contacts will no longer have any effect on the size of the conditioned reflex, but

will still be manifested in an increasing resistance to inhibitory influences.

What far-reaching consequences are involved in this statement it is easy to observe. For it follows that the stabilization of a conditioned reflex, measured by its degree of resistance to inhibitory influences, does not continue to an unlimited extent, but increases only as long as, in practice, new synaptic connexions do increase between the two concurrently activated centres. So the increasing resistance of the reflex to inhibitory factors may constitute a *measure* of the increase in synaptic connexions at that stage of conditioning when the reflex has already achieved its maximum value. But it must be pointed out that, if our arguments are correct, the discrepancy between the stabilization of a reflex (in the aspect now being considered) and the increase of its response should chiefly concern conditioned reflexes to strong stimuli. In reflexes to weak stimuli these two phenomena should proceed approximately parallel The performance of suitable experiments would make it possible to base the foregoing argumentation on more solid ground In the meantime it must remain a hypothesis.

3

Hitherto we have considered the magnitude of a conditioned reflex in relation to the increase in the number of synaptic connexions between the two centres concerned, i.e. as a function of the plastic changes in the cerebral cortex. Now we shall study the variability of reflexes in relation to the excitability of the centres engaged in them. This excitability can be varied either by acting specifically on those centres, or by acting pharmacologically on the entire nervous system.

There have been no detailed investigations into the relationship between the strength of the conditioned reflex and the excitability of the conditioned centre. On the other hand, investigations concerning the influence of the excitability of the unconditioned centre on conditioned reflexes have been numerous and manifold. Increase of excitability in the unconditioned alimentary centre has been achieved by fasting, and a decrease of its excitability by feeding the dog before the experi-

ment. The common result of all experiments of this type is that with a high alimentary excitability the conditioned reflexes rise, and with a low alimentary excitability the reflexes fall. This result is clear, and calls for no comment. However, another fact, obtained by the majority of the experimenters (Skipin, Dolin, Norkina and others*), calls for attention It is that with a diminution of alimentary excitability conditioned reflexes to weak stimuli fall much more than conditioned reflexes to strong stimuli. It often even happens that conditioned reflexes to weak stimuli drop to zero, whereas the effect of strong stimuli is hardly diminished.

This fact, which in the light of Pavlov's theory is rather unexpected, can be easily understood in the light of our preceding arguments A change in alimentary excitability induces a change in the 'characteristic' of the nerve cells of the alimentary centre, in the sense that with a diminution of excitability the curve *ABC* shifts to the right, but with heightened excitability it shifts to the left (fig. 6). As can be seen from the diagram, conditioned reflexes to weak and average stimuli undergo great changes as the result of these shifts, whereas a conditioned reflex to a strong stimulus remains in practice without change. For this stimulus ensures with excess a maximal response in all the effector units of the given centre, and so it can hardly be influenced by slight changes in the characteristic of that centre.

But we must point out that there are many unclarified features in the problem we are discussing. In the diagram (fig. 6) it is assumed that changes in the characteristic of the alimentary centre due to greater or lesser alimentary excitability touch only the threshold value of stimulation, and not the maximal response elicitable from this centre (OC'). Probably this assumption is correct only in the case of slight changes in alimentary excitability, whereas with more considerable changes it is possible that the maximal response also alters, and in consequence the curve *ABC* takes quite a different shape ($A_3B_3C_3$) In such a case, obviously, reflexes to both strong and weak

* Skipin, G. V., *Trudy Lab Pavlova*, 1941, vol x, p. 263; Dolin, A O, Personal communication, Norkina, L. N., Diss, 1944 (unpublished).

stimuli undergo changes. The whole problem presents an interesting and promising theme for experimental analysis.

A similar influence on the size of the reflexes is exerted by general changes in the excitability of the brain, caused by drugs. Small doses of caffein cause in general an increase in conditioned reflexes, small doses of narcotics cause their diminution; in this

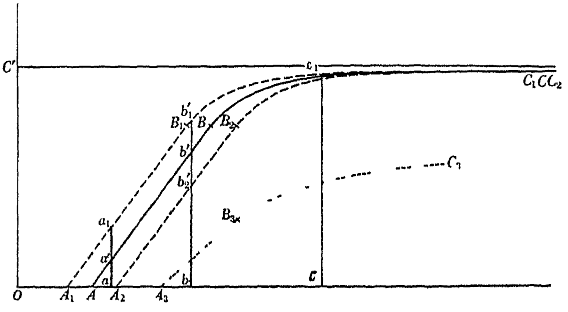


Fig. 6 Relation between the changes in the excitability of the alimentary centre and the size of conditioned reflexes ABC, the 'normal' characteristic of the alimentary centre neurons. $A_1B_1C_1$, the characteristic of alimentary centre in a state of increased excitability $A_2B_2C_2$, the characteristic of alimentary centre in a state of diminished excitability $A_3B_3C_3$, probable characteristic of alimentary centre in a state of strongly diminished excitability aa', the size of conditioned response to weak stimulus bb', the size of conditioned response to moderate stimulus. cc', the size of conditioned response to strong stimulus As is seen from the diagram, the alimentary excitability being diminished, the conditioned reflex to the weak stimulus becomes subliminal, to the moderate stimulus it is significantly reduced, and to the strong stimulus it remains practically unchanged The alimentary excitability being increased, the conditioned response to the weak stimulus increases greatly, to the moderate stimulus it increases less; to the strong stimulus it remains unchanged A very great diminution in alimentary excitability causes a significant decrease of all reflexes

latter case also, as Lebedinskaia's experiments show,* conditioned reflexes to weak stimuli undergo a greater fall than those to strong stimuli.

However, it must be added that the results of various experiments concerned with the influence of changes in the excitability of the nerve centres on the magnitude of the reflexes are highly

* Cf. Pavlov, *Conditioned Reflexes*, p. 279.

diverse, and often contradictory. This is due to the circumstance that in these experiments certain factors have not been taken into account. For instance, severe starvation of the animal results not in a heightening but a diminution of alimentary excitability, and, moreover, it causes general debility in the dog. Furthermore, it is necessary to allow for the fact that (as we shall see in the next chapter) strong conditioned stimuli cause also, to a greater or lesser degree, a defensive reaction. When the animal is in a normal state this reaction can be completely inhibited and imperceptible, but under the influence of stimulating drugs (such as caffein) or with a lessening of alimentary excitability, it may emerge more strongly, and distort the usual conditioned effect. This is perhaps the explanation of the fact that, under the influence of caffein, conditioned reflexes to strong stimuli sometimes suffer diminution,* and also the fact obtained by Rikman† (not, however, confirmed by other authors) that with a diminution in alimentary excitability strong stimuli sometimes lost more of their effectiveness than weak ones, and in their presence the dog refused to take the food.

Because of the above-discussed discrepancies we judge that all this field of phenomena calls for new, systematic and fundamental experimental study.

4

In our discussion so far on the problem of the formation of the conditioned reflex we have omitted one aspect which plays a great role in experimental work, and has far-reaching theoretical consequences.

As was emphasized in Chapter II, one of the indispensable conditions for the elaboration of a conditioned reflex is that the application of the stimulus to be conditioned should slightly precede the reinforcing stimulus. If the former stimulus starts to act when the unconditioned stimulus is already in action a stable conditioned reflex fails to be established, and an inhibitory

* Cf. Zimkin, N. V, *Trudy Lab Pavlova*, 1928, vol III/1, p 79, Zewald, L O, ibid. 1938, vol VIII, p. 369, Kleshchov, S. V., ibid 1938, vol. VIII, p. 182.

† Unpublished experiments Vide Pavlov, *Lectures*, vol II, pp. 54 et seq.

reflex arises in its place (see p. 19).* Leaving the discussion of this last point to one of our later chapters, we must now analyse the significance of the above-mentioned temporal order of the stimuli for the conditioning process.

The bringing into operation of the unconditioned stimulus connotes, of course, a sudden *rise* of activity of the corresponding centre. No matter what the state of this centre before the application of the stimulus, whether it was 'quiet' or subliminally excited, the action of the proper stimulus abruptly raises the excitation and makes it supraliminal. As we have just indicated, it is with this moment of its operation that a conditioned stimulus (or its traces) must coincide in order to bring about the formation of the normal conditioned reflex.

It follows from this that our statement in the foregoing chapter, that the conditioned reflex is formed when two coupled cortical centres are activated simultaneously, is slightly inaccurate. In reality the optimal (if not necessary) pre-condition for the formation of the conditioned reflex is the coincidence of excitation of the emitting centre with a sufficiently abrupt rise of activity in the receiving centre. In other words, in order to obtain a conditioned reflex of normal size and stability the impulses set up by the centre to be conditioned must reach the unconditioned centre in the stage when the latter's activation is growing. This inference will be given further support in later chapters, where we shall show that inhibitory conditioned reflexes arise, on the contrary, when excitation of the emitting centre coincides with the abrupt *fall* of active excitation of the receiving centre as the result of the cessation of action of the unconditioned stimulus, or of non-reinforcement of the conditioned stimulus by the unconditioned stimulus.

* Although single authors (both in U S S R and in America) claimed to have obtained positive conditioned reflexes with the reverse order of stimuli, the result of such a 'backward conditioning' (as it is called) was always uncertain and insignificant. Therefore the opinion now prevails that 'backward conditioning' does not exist at all, and that the positive effect sometimes obtained may be due to sensitization or pseudoconditioning (cf Hilgard, E R and Marquis, D. G , *Conditioning and Learning*, New York, 1940, p. 160).

If this principle is accepted, it becomes very easy to understand another well-known phenomenon, that of the formation of a conditioned reflex of the second order. It has long been recognized that if a stable and firmly established conditioned stimulus is from time to time preceded by another, indifferent stimulus (the conditioned stimulus not then being reinforced), then, if certain precautionary measures be observed, a conditioned reflex of the second order can be obtained. Such a reflex is formed especially readily when the conditioned stimulus of the second order is rather weak, while the conditioned reflex of the first order is strong and adequately fixed. In other cases, with such an experimental procedure an inhibitory reflex is established (conditioned inhibition, see p. 10).*

The explanation of this fact does not involve any difficulty. The excitation of the new stimulus coincides with the sudden rise of activity of the unconditioned centre produced by the application of the conditioned stimulus, and, therefore, it is not surprising that in such circumstances functional connexions arise between the centre of the new stimulus and the unconditioned centre The following fact obtained by Lindberg† is proof of the validity of this explanation After the elaboration of an alimentary conditioned reflex of the second order the conditioned reflex of the first order was transformed from an alimentary into a defensive one by replacing the food reinforcement with an electric shock applied to the paw. When this had been achieved, the action of the conditioned stimulus of the second order was tested It was found that it completely preserved its former significance, i.e. it remained a conditioned alimentary stimulus; which proves that the centre of this stimulus was indeed linked up with the alimentary centre, but not with the conditioned centre of the first order.

The linking up of a centre of a conditioned stimulus of the second order chiefly with an unconditioned centre does not, however, exclude the possibility of establishing connexions between the centres of the two 'indifferent' stimuli Thus, in the above-cited Narbutovich and Podkopayev experiments the

* Fursikov, D. S., *Trudy Lab Pavlova*, 1924, vol. i/1, p 3.
† Personal communication.

link between the simultaneously presented 'indifferent' stimuli (hissing and the sight of a rotating object) was so strong that when a conditioned defensive reflex to the rotating object was elaborated, the same reflex was formed spontaneously to hissing.* These data, which require both confirmation and further, more detailed, examination, can throw much new light on the mechanism of the formation of intercentral connexions.

* Vide Chapter v, § 5 Similar results were obtained by W J Brogden, *J. Exper Psychol*, 1939, vol xxv, p 323

Chapter VII

Alliances and antagonisms in the field of conditioned reflexes

(The summation of conditioned reflexes, and external inhibition)

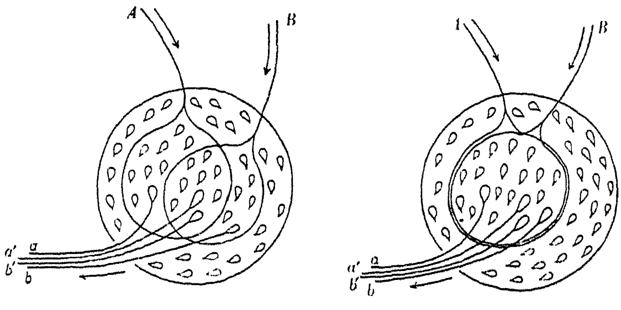
1

In the preceding chapter we discussed the basic properties of a *single* conditioned reflex; now we shall consider the laws of *interaction* of various reflexes.

It must be recalled that conditioned reflexes reinforced by one and the same unconditioned reflex are called *homogeneous* reflexes, and conditioned reflexes reinforced by different unconditioned reflexes are called *heterogeneous* reflexes. We shall call an assembly of homogeneous reflexes a *group of reflexes*, and for the sake of convenience the unconditioned reflex reinforcing the conditioned reflexes will also be referred to as appertaining to the same group. The unconditioned reflex will also be said to be homogeneous with all conditioned reflexes reinforced by it, but heterogeneous to all conditioned and unconditioned reflexes appertaining to other groups of reflexes

It can easily be seen that homogeneous reflexes are always allied, since, being reinforced by the same unconditioned stimulus, they have qualitatively exactly the same effect. Moreover, these reflexes represent an ideal form of alliance, such as is hardly found in lower reflexes. For whereas in the latter case different stimuli from the receptor field of the given 'type reflex' always evoke a somewhat different configuration of excitations and inhibitions in the nervous system, and their effect is never quite identical, in the case of homogeneous conditioned reflexes the neuron fields corresponding to various conditioned stimuli are connected with one and the same unconditioned centre, and so the convergence of the given group of conditioned reflexes is always complete. These relations are diagrammatically presented in fig. 7.

Comparatively little attention has been given by the Pavlov school to the problem of summation of conditioned reflexes, since in his theoretical system this problem was of rather secondary importance. That is why in our exposition of the main principles of his theory the discussion of this problem was omitted. From experiments concerning summation* conducted by the Pavlov school it follows that if one at least of the two



(a) (b)

Fig 7. The partial convergence of the majority of spinal allied reflexes (*a*) as compared with the total convergence of conditioned homogeneous reflexes (*b*) *A*, *B*, two afferent nerve paths *a*, *a'*, *b'*, *b*, efferent nerve paths. More detailed explanations will be found in the text Fig. 7*a* is taken from Sherrington, *Proc Roy Soc* B, 1929, vol cv, p. 332.

concurrently acting conditioned reflexes possesses top value when elicited separately, their joint action also gives a top effect, though in Rikman's experiments (not confirmed by others) sometimes the effect was rather less When both the reflexes acting separately have a value exceeding half the top value, their joint action also gives a top effect. But when both reflexes are still weaker, then, as Pavlov claims, the conjoint effect is approximately the exact sum of their separate responses.

It is noteworthy that although the Sherringtonian interpretation of the foregoing facts quite obviously fits them perfectly, in this case also Pavlov remained faithful to his own conception. Certainly he did admit that 'the results of summation of weak

* Rikman, V. V , *Trudy Lab Pavlova*, 1928, vol II/2, p 13; Yakovleva, V. V., ibid 1932, vol. IV, p. 268, Galperin, S I , ibid. 1940, vol. IX, p. 5, Zewald, L. O , ibid, 1940, vol. IX, p. 62, and others.

conditioned stimuli might naturally be regarded as the fusion of the effect of both weak stimuli in the point of the cortex with which, in all these particular experiments, the conditioned stimuli are brought into relation, viz. the chemical analyser in the cortex'. 'But', he argues further, 'on the other hand the summation of the weak conditioned stimulus with the strong, and of the two strong together, definitely *points to the cells pertaining to the conditioned stimuli themselves*. We have every right to regard the interrelation of processes in summation as taking place somewhere *within the above-mentioned sets of cells*.'* So it is not surprising that when summation is treated from this viewpoint, phenomena not particularly difficult to understand have 'hitherto not lent themselves to solution'.

Analysis of the data given above from the aspect of the laws of summation of allied reflexes presents no difficulty (cf. fig. 3). It is easy to see that in the interaction of two strong conditioned stimuli the phenomenon of occlusion arises In fact, if one of the conditioned stimuli evokes a top conditioned reaction (which is defined by the magnitude of the field of excited cells in the unconditioned centre and by the maximal effect elicited from each cell), the second stimulus cannot add anything to this reaction. In this respect, as we have pointed out, homogeneous conditioned reflexes differ from ordinary allied reflexes, in which a singular stimulus is only exceptionally capable of eliciting a response of maximal size for the reflexes of the given group. This is explained by the relative convergence existing in the majority of allied inborn reflexes, as contrasted with the absolute convergence existing in homogeneous conditioned reflexes.

When both the concurrent conditioned stimuli are not so strong, there is less occlusion, and therefore the summation of the responses of the two reflexes can be observed

From the above summary it is evident that in the experiments of the Pavlov school the case in which both the concurrent conditioned stimuli are so weak that the phenomenon of facilitation can arise in their joint effect was overlooked. We have filled this hiatus, and as we have not previously published the results obtained, we summarize them here.

* Pavlov, *Lectures*, vol. I, p. 388 (our emphasis).

	Conditioned stimulus 1	Its effect	Conditioned stimulus 2	Its effect	Joint effect of both stimuli	Maximum value of reflexes on the given day
1st dog:	Lamp	38	Touch	33	66	66
	Lamp	40	Metronome	61	65	65
	Lamp	39	Metronome	59	59	59
	Very dim lamp	18	Continuous touch	15	37	57
	Very dim lamp	18	Continuous touch	29	64	70
2nd dog.	Metronome	42	Touch	40	53	53
	Metronome	47	Touch	33	55	55
	Very dim lamp	8	Continuous touch	16	48	50
*	Very dim lamp	3	Continuous touch	2	29	29

* Dog fed before experiment

Note In both dogs conditioned alimentary reflexes to the above-presented stimuli were established The isolated period of the conditioned stimuli lasted 30 sec The numbers given in the table express the quantity of conditionally secreted saliva in scale graduations (one graduation = 0 01 cm ^{3})

The above table shows that the result of concurrent application of strong and weak stimuli exactly corresponds to the results obtained by other authors. But in regard to the concurrence of very weak stimuli (such as the dim lamp lit in a lighted room, and the continuous tactile stimulation of a dog's unshaven skin) an outstanding facilitation of both reflexes is obtained ('18' + '29' = '64' in one dog, and '8' + '16' = '48' and '3' + '2' = '29' in the other).

The last row of figures given in the table calls particularly for attention. For three or two graduations of saliva secreted in 30 seconds is really almost zero effect. So we obtained a result in which each of the separately acting conditioned reflexes was really subliminal (not in the sense of action on the corresponding receptors or centres of the given analyser, but in the sense of action on the unconditioned centre), but in sum they yielded a noteworthy conditioned response

In view of the importance of this result we quote the report on the experiment in its entirety. The dog was given a good meal before the experiment.

Time in min	Conditioned stimulus	Isolated action of stimulus sec	Secretion of saliva per 10 sec	Total secretion	Reinforcement
1	Very dim lamp	30	0, 0, 4	4	Food
6	Metronome	30	8, 7, 14	29	Food
10	Very weak touch	30	0, 0, 0	0	Food
15	Very dim lamp	30	3, 0, 0	3	Food
20	*Very dim lamp and very weak touch*	30	5, 11, 13	29	Food
26	Metronome	30	4, 0, 11	15	Food
31	Very weak touch	30	0, 0, 3	3	Food
35	Metronome	30	0, 12, 13	25	Food

As can be seen from this report, each of the very weak stimuli, applied separately, evoked hardly any secretory reaction, but applied in association they produced the top response for that day. But it must be pointed out that experiments concerning the summation of responses to very weak stimuli come up against one fundamental difficulty, namely, the extraordinarily marked fluctuation in the size of the conditioned reflexes to these stimuli. For instance, the same stimulus in the same experiment may cause a minimal conditioned reaction at one time, and a quite imposing reaction, equal approximately to half the top value, at another. It is not difficult to explain this variability. In every dog submitted to stereotyped conditioned reflex experiments a subliminal conditioned reflex 'to time' is established; this reflex easily summates with reflexes to other weak stimuli, resulting in considerable facilitation, whereas, because of occlusion, it has only insignificant influence on the effect of strong stimuli. In other words, the variability of reflexes to weak stimuli and their comparative constancy to stronger stimuli are an expression of those very laws of summation which we are now discussing.

It is worth pointing out that, as Cooper, Denny Brown and Sherrington report,* the same kind of difficulty is encountered when the summation of weak spinal reflexes is investigated. Here are their words: 'But such results (i.e. facilitation) although fairly frequent, have not been of regular occurrence, and reflex contractions in response to stimuli not far above threshold are wont to be irregular in size. The control observations as gauge

* *Proc. Roy. Soc.* B, 1927, vol. CI, p. 262.

and standard for the size of the interacting concurrent reflexes are for these low values of stimulus much less trustworthy.' This quotation is striking evidence of the harmony that exists between the properties of conditioned and inborn reflexes even in regard to such tiny details.

2

Now we turn to discussion of the interaction between heterogeneous reflexes. It has been usual to investigate this interaction on the basis of the effect exerted by so-called extraneous stimuli (i.e stimuli causing an heterogeneous unconditioned reaction) on the conditioned reflex; and generally for this purpose stimuli eliciting an orientation reaction have been chosen. In connexion with the inhibitory action of such stimuli on conditioned reflexes the concept of external inhibition came into being, and later that of negative induction (p. 19).

It is obvious that external inhibition can be completely explained from the viewpoint of the general laws governing reflex activity, and that it is nothing but one of the numerous manifestations of interference between antagonistic reflexes. In fact, as we pointed out in Chapter IV, each reflex (conditioned or unconditioned) always involves a great number of effectors, and affects the whole nervous system in one way or another. Thus, if one of the concurrently operating reflexes causes excitation (supra- or subliminal) in certain groups of neurons, and inhibition in other groups, while another reflex causes excitation of those groups which in the former reflex were inhibited, and inhibition of those groups which were formerly excited, the result of their joint action will be a mutual weakening of both the reflexes. The degree of this diminution depends on the strength of the two stimuli, and on the excitability of the nerve centres involved.

It has to be mentioned that while the supraliminal excitation of any group of neurons is a phenomenon directly observable and measurable, subliminal excitation and also inhibition do not manifest themselves directly, and can be detected only *with the aid* of the action of other reflexes, reflexes allied with the given reflex make it possible to detect the concealed excitation of

certain groups of cells, while antagonistic reflexes do the same for inhibition. For instance, in eliciting an alimentary reflex alone (whether conditioned or unconditioned) it is possible to recognize only the excitation of those groups of neurons which are excited supraliminally, i.e. which form the so-called alimentary centre. But it is not known what happens meanwhile in other centres, e.g in the centres of various defensive reflexes, of the sexual reflex, etc. In order to have evidence on this question an extraneous reflex must be elicited concurrently with the alimentary reflex, and the increase or decrease of its (the extraneous reflex) response will indicate whether the corresponding centre is subliminally excited or inhibited by the alimentary stimulus. Conversely, the changes in the alimentary reflex will provide evidence of what is happening in the alimentary centre during the operation of one or another heterogeneous reflex.

It is worth mentioning that this is the same method that was so successfully applied more than 40 years ago by Sherrington in his investigations into the integrative action of the spinal cord, and which enabled him to elucidate the actual structure of many spinal reflexes.

Unfortunately, the problem of interaction between heterogeneous reflexes has been investigated by the Pavlov school very inadequately. Apart from numerous and detailed works on the influence on alimentary and acid conditioned reflexes of stimuli evoking an orientation reaction, there have been hardly any investigations into the mutual interaction of other pairs of reflexes. And so this extensive field so far represents an almost unchartered province of physiology of the higher nervous activity, and only a few fortuitous observations point to its far-reaching importance and interest.

In Chapter II we referred to Bezbokaya's experiments, in which an animal's aggressive reaction caused an increase of the alimentary conditioned reflexes, which Pavlov explained as an irradiation of strong excitation from the centre of aggression throughout the nervous system. In our view this fact is to be explained as follows· Between the reflex of aggression and the alimentary reflex (perhaps in connexion with their biological

affinity) there is a concealed alliance, expressed in the fact that in a state of aggression the alimentary centre is subliminally excited. This excitation, summating with a supraliminal excitation of this centre, evoked by an alimentary conditioned stimulus, causes an increase of the conditioned response. It is a pity the experimenter did not examine how the aggression acted on acid or other defensive conditioned reflexes; that would have made it possible to estimate whether it was a case of unspecified irradiation of excitation, or a definite, purely selective summation of corresponding reflexes.

A result similar to that of Bezbokaya's experiments was obtained by Bieliakov.* This author employed as extraneous stimulus a trumpet of unusual and piercing sound, which provoked in the dog a violent aggressive reaction in the form of barking, aggressive movements, etc. This stimulus caused a complete disinhibition of the inhibitory alimentary reflex elicited after it As we shall see later, this is the result of the summation of the subliminal excitation of the alimentary centre, evoked by the inhibitory stimulus, with the subliminal excitation of this centre, caused by the stimulus of aggressive reaction (vide Chapter X) It is worth while recalling here that strong extraneous stimuli, causing not an aggressive but an orientation reaction of a passively defensive character, not only do not disinhibit the inhibitory reflexes, but, on the contrary, inhibit active alimentary reflexes, both conditioned and unconditioned.

So, as can be seen from these experiments, aggressive reflexes enter into a partial alliance with alimentary reflexes, as they both cause an excitation in the alimentary centre; but, while in the alimentary reflex this excitation is manifest, in an aggressive reflex it is subliminal and can be detected only by aid of the former reflex. It is a matter for future investigations to discover how the relations between these two reflexes in the field of other effectors, and in animals other than carnivorous, are arranged.

Similar relations of a partial alliance are also to be observed in heterogeneous defensive reflexes As we shall show in the next section, an alliance exists between the defensive reflex against

* Bieliakov, V. V , Diss , Petersburg, 1911 (Pavlov, *Lectures*, vol. I, pp. 172–3).

the introduction of acid into the mouth and the defensive reflex to stimulation of the paw with an electric shock (or between corresponding conditioned reflexes); this alliance consists in the acid reflex strengthening the local defensive reaction against the stimulation of the paw and, on the other hand, the defensive reflex to the electrical shock causing an intensification of the defensive reaction against the introduction of acid.

The facts cited wholly confirm the thesis formulated by Sherrington 40 years ago that each particular reflex has a much more compound structure than one could have expected if only their observable effects had been taken into account, and that this structure can be elucidated by investigation of the results of the simultaneous action of different reflexes. Although systematic investigations of this type have not yet been undertaken in regard to higher reflexes, the fortuitous data available indicate that definite groups of reflexes (e.g. alimentary and aggressive reflexes on the one hand, various defensive reflexes on the other) enter into partial alliance with one another, i.e. they cause partially identical excitato-inhibitory patterns in the nervous system Future research should clear up more precisely the essence of these common elements of different reflexes, and should thus provide a basis for proper classification of animals' reflex activity.

3

So far we have been speaking of alliances and antagonisms between reflexes, whether conditioned or unconditioned, elicited by two separate stimuli. Study of the interrelations between such reflexes is a simple extension and complement of investigations already carried out in the field of spinal and subcortical reflexes. But it has to be pointed out that conditioned reflex activity provides yet another very interesting possibility of investigating interrelations between heterogeneous reflexes, one not met with in the field of ordinary reflexes, and having certain advantages over the method already described.

Let us assume that there are two unconditioned reflexes, $S_1 \rightarrow R_1$ and $S_2 \rightarrow R_2$, and that, by applying the stimuli S_1 and S_2 in overlapping sequence a number of times, the first becomes

conditioned and begins to produce the reaction R_2. Thus the stimulus S_1 becomes the source of two reflexes: 'its own' unconditioned reflex $S_1 \rightarrow R_1$, and the new, imposed conditioned reflex, $S_1 \dashrightarrow R_2$. These two reflexes may be either in alliance or in antagonism with each other, and the results of the experiment will depend on which of these alternatives applies.

Let us first consider these two possibilities from the theoretical aspect, utilizing the diagram in fig. 8.

C_1 and C_2 are two 'unconditioned centres', and in order to avoid complicating the diagram and making our analysis difficult we treat each of them as a whole, not distinguishing their respective parts (e.g. the receptive centre of the unconditioned stimulus and the executive centre, the cortical and the subcortical parts of these centres, etc.). We take into account only the unconditioned reactions R_1 and R_2, ignoring all others.

Fig. 8*a* represents the case in which the two centres are in antagonistic relations, i.e. the activation of either of them produces the inhibition of the other. If the stimuli S_1 and S_2 are applied in overlapping sequence, conditioned connexions are formed from the centre C_1 to the centre C_2. The conditioned reflex thus produced will be elaborated slowly and laboriously, since the excitatory conditioned connexions must become numerous enough to preponderate over the unconditioned inhibitory connexions already existing between the two centres. When this reflex is formed the situation will be as follows: the stimulus S_1 will produce an excitation of the centre C_1, which because of the conditioned connexions will cause an excitation of the centre C_2; the excitation of this latter will exert inhibitory influence on the centre C_1; as the result the stimulus S_1 will produce the reaction R_2, but this reaction will always remain 'stunted', because of the strong admixture of inhibitory impulses travelling along the path i_1, simultaneously its own reaction also will be more or less inhibited because of the inhibitory impulses coming from the centre C_2 to the centre C_1 along the path i_2. So in this case the conditioned reflex $S_1 \dashrightarrow R_2$ will be constantly inhibited by the unconditioned reflex $S_1 \rightarrow R_1$, while this latter also will be inhibited by the conditioned reflex $S_1 \dashrightarrow R_2$.

Fig. 8*b* represents the case when the centres C_1 and C_2 are in a state of concealed alliance. Let us apply the stimuli S_1 and S_2 so as to form conditioned connexions from C_1 to C_2. The conditioned reflex thus obtained will be elaborated readily and rapidly, and will be of considerable magnitude, since the excitatory impulses travelling along the conditioned connexions co_1 will summate with the excitatory impulses travelling along the

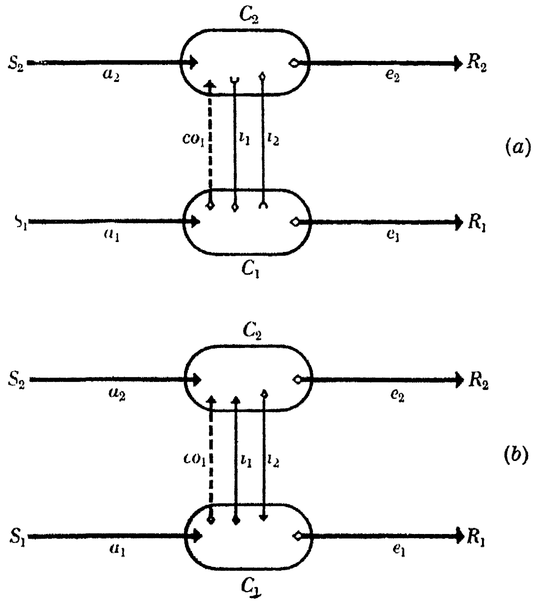


Fig. 8. The interrelations between the cortical 'centres' (C_1 and C_2) when repetitively excited in overlapping sequence Stimulus S_1, precurrent; stimulus S_2, postcurrent ⟶ Excitatory inborn connexions. ---→ Excitatory acquired connexions ——C Inhibitory inborn connexions a_1, a_2, afferent pathways e_1, e_2, efferent pathways i_1, i_2, inborn interconnexions between the centres. co_1, conditioned connexion between the centres. (*a*) Reflexes $S_1 \rightarrow R_1$ and $S_2 \rightarrow R_2$ are antagonistic After conditioning is established stimulus S_1 evokes an excitation of centre C_2 through co_1, stunted by inhibitory impulses travelling through i_1. The centre C_1 is also inhibited by impulses travelling along i_2 (*b*) Reflexes $S_1 \rightarrow R_1$ and $S_2 \rightarrow R_2$ are allied After conditioning is established stimulus S_1 evokes an excitation of centre C_2 (through co_1) enhanced by excitatory impulses travelling along i_1. The excitation of the centre C_1 is also enhanced by excitatory impulses travelling along i_2 Further explanations in text.

unconditioned connexions i_1. The result will be as follows: the stimulus S_1 will evoke excitation in the centre C_1, which will cross to the centre C_2 along the paths co_1 and i_1, and consequently will initiate the reaction R_2. But simultaneously the stimulus S_1 will produce its own reaction R_1, which will be stronger than before, since it will be strengthened by the impulses coming from the excited centre C_2 along the paths i_2 Thus in this case the reflex $S_1 \dashrightarrow R_2$ will not only be enhanced by the reflex $S_1 \rightarrow R_1$, but will also have facilitating influence on it.

The existing experimental material strikingly confirms the foregoing considerations

The case presented in fig. 8*a* is obtained when the stimulation of the skin by an electric shock (S_1) is reinforced by food (S_2). As the defensive and alimentary reflexes are antagonistic to each other, the following situation arises: (1) the conditioned alimentary reflex to stimulation by electric shock is formed slowly and laboriously; (2) this reflex is always more or less stunted, never reaching the magnitude of conditioned reflexes to strong stimuli, (3) the unconditioned defensive reflex ($S_1 \rightarrow R_1$) is completely or almost completely suppressed.

A quite different state of affairs is observable in the case when stimulation of the skin with an electric shock is reinforced by introduction of acid into the dog's mouth. Only in one short series of experiments performed by Fiodorov* has this subject been investigated, and unfortunately the results have not been adequately analysed; none the less they are sufficient to provide a demonstrative confirmation of our previous argument. These experiments yield evidence that when stimulation of the skin with an electric shock was reinforced by introducing acid into the mouth, the defensive reaction against shock not only did not diminish, but, on the contrary, increased greatly, the dog tried to tear away the electrodes with his mouth, developed strong motor excitement, and so on. Despite the existence of this reaction the *conditioned* reflex to stimulation with electric shock, directed against the introduction of acid ($S_1 \dashrightarrow R_2$), was formed very rapidly, and soon reached a greater value than to

* Fiodorov, V. K., *Trudy Lab. Pavlova*, 1933, vol v, p. 199.

other stimuli. It would be difficult to have a more obvious example of mutual facilitation between two reflexes.

Another, no less interesting fact of the same kind was obtained by Pietrova.* This investigator reinforced a rhythmic tactile stimulus acting on a dog's hindleg by introducing acid into the mouth. When the conditioned reflex was formed it was found that the tactile stimulus, which previously had caused no defensive reaction, began to evoke a rhythmic defensive flexion of the limb in time with it. This fact, at first glance very strange, is easily understood when considered in the light of the foregoing argument. The rhythmic tactile stimulus of the leg undoubtedly provokes a weak subliminal defensive reaction, which in normal conditions, or when the tactile stimulus is reinforced by food, is not manifested at all. But when this stimulus is reinforced by introduction of acid, i.e. a stimulus itself producing a defensive reaction, the local defensive reaction against the rhythmic touching is facilitated and becomes manifest.

With the help of the relations above described it is also possible to explain the properties of alimentary conditioned reflexes to the so-called supramaximal stimuli. As will be recalled from Chapter II, the properties of these reflexes gave rise to the conception of 'the top of capability of cortical cells' and of 'top inhibition', which was used to explain the low value of the conditioned response to this kind of stimuli. As we showed in Chapter III (§ 3), this explanation will not withstand criticism, and cannot be accepted. Now we can prove that it is completely unnecessary.

There is not the least doubt that a supramaximal stimulus causes a strong defensive reaction in a dog (as in human beings), which is displayed quite clearly: it shivers, runs from the stand, or refuses to jump on to it, will not take food, etc. So it is not surprising that if such a stimulus is reinforced by food, it becomes the source of two antagonistic reactions: defensive and alimentary. The situation that arises in consequence is shown in fig. 8*a*. Though the defensive reflex $S_1 \rightarrow R_1$ is more or less suppressed, the conditioned reflex $S_1 \dashrightarrow R_2$ is formed with difficulty and remains stunted.

* Pietrova, M. K., Diss., Petersburg, 1914.

The more timid the dog, i.e. the stronger the defensive reaction evoked by the supramaximal stimulus, the more difficult it is for the alimentary reflex to overwhelm it, and so the alimentary conditioned reflex is formed with particular difficulty and remains particularly small. So the phenomenon that Pavlov calls a low top of capability of the cortical cells, and which he attributes to 'the weak type of an animal's nervous system', we ascribe to the animal's timidity, i.e. to the increased excitability of its defensive centres. The factors that further increase this timidity also make it difficult to elaborate an alimentary conditioned reflex to a supramaximal stimulus, and, conversely, factors which enhance the alimentary excitability give this reflex relative preponderance.

It is also easy to understand that a supramaximal stimulus introduced into the stereotype of conditioned reflexes (vide Chapter XI) disturbs all the conditioned reflex activity sometimes for several days. This is because the whole experimental situation gets conditioned to the supramaximal stimulus, and itself tends to elicit the defensive reaction. This explains all the disorders in conditioned reflexes and in the animal's behaviour during the experiment, which gradually disappear when the supramaximal stimulus is withheld.

In connexion with this interpretation of the nature of supramaximal conditioned stimuli, important new problems arise which the Pavlov theory did not consider at all. What will be the magnitude of the conditioned reflex to a supramaximal stimulus reinforced by the introduction of acid into a dog's mouth, or by the stimulation of the paw with an electric shock? Will the conditioned stimuli then also be smaller than to other stimuli, or, on the contrary, will the effect of these reflexes be increased because of the summation of the two defensive reflexes, conditioned and unconditioned? Investigation along these lines would render it possible to obtain more detailed knowledge of the structure of the defensive reflex to supramaximal stimuli, and would enable us to abandon the unfortunate conception of top inhibition altogether.

When speaking of the summation of conditioned reflexes to strong stimuli, we drew attention to the results of Rikman's

experiments, in which the joint effect of the action of such stimuli was occasionally less than that of each stimulus separately. Though this fact has not been confirmed by other investigators, it seems to be quite possible. In fact, if each of the strong conditioned alimentary stimuli evokes a subliminal defensive reaction, which normally does not influence the conditioned reflex, when they act jointly both defensive reactions are mutually facilitated so much as to bring about a manifest inhibition of the alimentary reflex with which they are in antagonism.*

While alimentary conditioned reflexes to supramaximal or other stimuli, evoking a defensive reaction, are formed with some difficulty, on the other hand they are formed rapidly and easily to stimuli of which the reaction is in concealed alliance with the alimentary reflex. The experiments conducted by Cytovich may serve as an example of this phenomenon.† In his well-known experiments Cytovich has shown that in the dog the alimentary reflex to the smell of meat is not an unconditioned but a conditioned reflex. In order to demonstrate this fact he fed the dogs for a long time after birth exclusively on milk. When the animals were shown meat for the first time no salivation was evoked either by its sight or its smell. Only when the dogs had been fed with meat once or twice did the smell of this food start to evoke the salivary reaction.

Although the fact described by Cytovich seems to be quite convincing, reading his paper one is struck by the circumstance that from the very beginning the smell of meat was very attractive to the dogs (they sniffed at it, and tried to seize it with the

* The following situation which arose in Rikman's experiments is worthy of attention. In his experiments on alimentary excitability (of which we have spoken in the previous chapter) he found that, despite the evidence of other authors, in a state of a reduced alimentary excitability the conditioned reflexes to strong stimuli diminished more than those to weak stimuli. In the experiments with summation now cited, again, despite the evidence of other authors, two strong stimuli given in association caused a lesser effect than each of the two separately. From this should it not be deduced that in these experiments, either as the result of the individual properties of the dogs or as the result of the particularly strong sound of the auditory stimuli used, these stimuli brought out a concealed defensive reaction which became manifest in circumstances favourable to it, such as were supplied by the experiments described?

† Cytovich, I. S., Diss., Petersburg, 1911.

mouth) and that the elaboration of the conditioned reflex to the smell of meat occurred extraordinarily rapidly, much more rapidly than it usually occurs to other 'indifferent' stimuli. In our view this fact must be considered as showing that between the inborn reflex to the smell of meat (contrary to the inborn reflexes to the smell of some repellent substances) and the alimentary reflex a concealed alliance exists, which is manifested by the very rapid and easy formation of the corresponding conditioned reflex and by its strength and stability.

From all the above data it follows that the cerebral cortex cannot be considered as a uniform structure capable with equal facility of putting through any intercentral connexions, like an automatic telephone exchange, but that it possesses a definite organization, which makes the formation of certain connexions an easy and rapid process, and others, on the other hand, difficult and complex We may expect that suitable experimental investigations undertaken in this direction will throw more light on this problem.

All these facts show how erroneous was Pavlov's view that the relative strength of the two concurrently applied stimuli was the factor which determined the direction of conditioning in the sense that 'every strongly excited centre *attracts* to itself every other weaker excitation'. As we have argued in the preceding chapter, the direction of conditioning depends primarily upon the temporal succession of two concurrently applied stimuli, the precurrent stimulus being conditioned, and the postcurrent stimulus conditioning. If, for instance, an electrical shock to the paw is conditioned to the introduction of acid into the animal's mouth (as in Fiodorov's experiments), it is not because this latter stimulus is stronger, but because the former was the 'leading' stimulus. We have no doubt that if the temporal succession of these stimuli were reversed, the introduction of acid into the mouth would evoke (besides its own reaction) conditioned flexion.*

* Although experiments of this kind with the use of acid have not been performed, we have experimental evidence to show that the introduction of sugar into the mouth can be conditioned to the electric shock (Savich, A , Diss., Petersburg, 1913)

It is true that in some cases the relative strength of the stimuli does play some role in the process of conditioning, a role which can be easily foreseen in the light of our foregoing discussion In fact, if two stimuli applied in overlapping sequence evoke antagonistic reflexes to one another, and the leading reflex is much more powerful than the subsequent reflex, then this latter reflex can be inhibited to such an extent by the former that the corresponding centre will fail to be activated altogether. In such a situation the elaboration of an active conditioned reflex to the precurrent stimulus (the post-current stimulus reinforcing) will be completely prevented. On the contrary, as will be seen in Chapter IX (§ 1), such a coincidence of stimuli results in the formation of an inhibitory conditioned reflex to the subsequent stimulus, with the cessation of the leading stimulus acting as a negative reinforcement.

Chapter VIII

The generalization of conditioned reflexes

The generalization of the conditioned stimulus would at first glance seem to be a phenomenon most easily interpreted in terms of irradiation of excitation. Indeed, if it is found that the formation of the conditioned reflex to stimulation of a given place on the dog's skin leads to the establishment of the same reflex to stimulation of other places, one is greatly inclined to think of irradiation of excitation as the physiological mechanism responsible for this fact. The evidence that irradiation of excitation really occurs in such cases as Jacksonian epilepsy and direct stimulation of the cortex* seems to support this view. On the other hand, however, at the present moment it is difficult to decide whether these phenomena can be regarded as the real prototype of normal cortical activity, or whether they represent rather exceptional events occurring only with the use of very strong stimulation applied directly to the cortical cells.

There is no doubt that the problem of generalization will find its final solution only when we ascertain what is the structure of 'the centre of the conditioned stimulus', and what is the intimate mechanism of its activity. Therefore we shall here confine ourselves only to presenting the arguments which dispose us to reject the Pavlovian conception of generalization based on irradiation of excitation, and to putting forward another conception which seems more adequate as an explanation of this phenomenon.

As is well known, the generalization of the conditioned stimulus extends to all stimuli *similar* to it, and the closer the similarity, the stronger is the generalization. But concretely all the cases of similarity between stimuli and, consequently, of the generalization of the conditioned reflex can be divided into the following three categories

(1) Stimuli are similar (i.e lead to generalization, if one of them is a conditioned stimulus) when they act differently on the

* Adrian, E D, *J Physiol* 1937, vol LXXXVIII, p. 127.

same receptors. This group includes, e.g. the differing frequency of beat of a metronome, the rhythmic stimulation of one and the same place on the skin with touches of different frequency, tones of the same pitch, but of different strength, etc.

(2) Stimuli are similar when the receptor fields excited by them partially overlap. This includes tones of the same pitch but different overtones, chords, possessing common tones, and generally all kinds of compound stimuli possessing certain components in common.

(3) Stimuli are similar when they excite different points of the same receptor surface in the same way. This includes the stimulation of the same kind applied to different areas of the skin, tones of different pitch played on the same instrument, etc

It is easy to observe that to understand the generalization of conditioned reflexes to stimuli belonging to the first two categories we do not need to postulate the existence of irradiation of excitation at all, unless it is accepted that, for instance, the beat of a metronome at different frequencies excites different cortical 'points'. On the other hand, the generalization of the third category of similar stimuli can easily be explained by the irradiation of excitation, if it is accepted, as one has every right to do, that there is 'point-to-point correspondence' between the given receptive surface and the corresponding area of the cortex.

But it must be observed that in experimental practice there is no difference whatever between these three categories of similar stimuli, and that the properties of generalization are absolutely identical, irrespective of whether it is a question of similar stimuli differing from one another in rhythm, or in particular elements, or in their position within the receptive surface. For instance, in all these cases there is a definite 'gradient' of generalization, as the result of which, the farther the stimulus is from the original conditioned stimulus (whether in the sense of rhythm, or common elements, or physical distance) the weaker is its effect; if one of the similar stimuli is not reinforced, differentiation occurs, and its 'difficulty' depends on the similarity between the two stimuli, etc. These data incline us rather to accept a single common principle for the explanation of generalization, since it is difficult to assume that this pheno-

menon would in different cases possess a completely different mechanism, which fails to be revealed in any experimental facts.

So, in order to interpret all the phenomena of generalization in a uniform manner by reference to a single principle, we have to accept one of the following postulates. Either it must be assumed that each individual stimulus gives rise to excitation in some 'point' (or limited group of cells) in the cerebral cortex, that the more similar the stimuli, the closer are the corresponding cortical points situated to one another, and that the excitation arising in any cortical point irradiates to neighbouring points, this constituting the source of generalization Or it must be assumed that the cortical centres of particular stimuli represent complex and widely dispersed formations, that they can partially overlap, and that this partial overlapping is the cause of generalization.

The conception of overlapping between cortical centres has many adherents among both physiologists and neurologists, and is supported by strong evidence in various fields of experimental data To begin with, as morphological evidence indicates, each point of the receptive surface has a corresponding large area of representation in the cerebral cortex, this being because of the extensive ramifications of the afferent nerve paths leading from the periphery to the centres The study of the action potentials developed in the cortex as the result of peripheral stimulation seems to point in the same direction Although in the deeply narcotized animal the stimulation of small receptive areas elicits electrical changes limited only to small zones in the cortex, the more the animal is awakened, the more complex and extensive these changes become.* Further, we must bear in mind that the 'cortical centre' of a given stimulus is not confined only to the group of cells receiving the impulses from the periphery, but includes also other elements situated both in the primary receptive fields and in 'associative' areas. The extensive evidence of so-called physiological neuronography fully confirms this view. Finally, it must be emphasized that stimuli dealt

* Vide, for example, Marshall, W. H , Woolsey, C N. and Bard, P , *J. Neurophysiol* 1941, vol IV, p. 1.

with in conditioned reflex study are always compound, which connotes that their cortical centres are of even greater complexity.

All these considerations strongly support the view that the 'centre of a stimulus' must be regarded as a broad multistoried and highly organized aggregate of cells, and that even if 'point-to-point correspondence' between the centre and periphery can be detected in various analysers, it may concern only the cortical 'entrance gates' of the particular simple stimulus. But it would be quite incorrect to regard such a single 'point' as the entire cortical centre of the given stimulus, responsible for all its complex properties.

The view just presented allows us to accept the assumption that the intimate nature of the phenomenon of similarity between various stimuli consists in the partial overlapping of the corresponding cortical centres. The more extensive the overlapping, the closer is the similarity between the respective stimuli, and when this overlapping is virtually complete the similarity passes into 'identity' Beyond all doubt this mechanism of similarity exists in the case of similar stimuli described as belonging to the second category above. For in this category individual similar stimuli partially overlap already at the level of the receptors, so there is no reason to doubt that these same relations exist in the centres. As for the first category of similar stimuli, i.e. those concerned with stimulation of the same receptors with different strength or frequency, in their case also it can be accepted that the corresponding cortical centres partially overlap. Such an assumption was actually adopted in Chapter VI in order to explain the dependence of the magnitude of the reflex response upon the strength of the stimulation. So there remains only the third category of similar stimuli, that in which individual stimuli affect quite *different* receptors. We assume that in this case also the centres of these stimuli partially overlap, and the more so, the closer the positions of the stimulated points, i.e. that the relations which in the case of the first two categories exist at the level of the receptors, here occur only at the level of the nerve centres (whether in the thalamus or in the cortex).

It is easy to see that the conception we have just presented quite satisfactorily explains all the properties of generalization.

First, it is quite clear that the more elements in common possessed by the centres of two stimuli the stronger will be the generalization if one of these stimuli has been conditioned. Hence the existence of a gradient of generalization can be deduced.

Further, it is known that the first application of a stimulus similar to the original conditioned stimulus evokes an abnormally low conditioned reaction, which on its repeated applications increases even when it has not been reinforced.* This fact is explained by the circumstance that the excitation of a part of the centre of the new stimulus which does not overlap with the centre of the conditioned stimulus causes an orientation reaction, which inhibits the conditioned reflex evoked by the excitation of the common part of both centres. This reaction is quickly rendered extinct, and in consequence the conditioned reflex to the similar stimulus is revealed in all its true strength

The extinction to zero of the primary conditioned reflex causes the complete extinction of reflexes to stimuli similar to it; this is obvious, since these reflexes are evoked by the excitation of those elements common with the elements of the conditioned centre. But the extinction of a reflex to a secondary conditioned stimulus causes only a partial extinction of the reflex to the original stimulus (the weaker, the more distant the stimulus), since the centre of the original stimulus contains elements which did not participate in the process of extinction.

Finally, it must be observed that the evidence provided in the previous chapter concerning subliminal conditioned reflexes (§ 1) compels us to recognize the existence of latent generalization when the given stimulus, having little similarity with the conditioned stimulus, evokes a subliminal conditioned response which can be detected only through its summation with other allied reflexes. We emphasize the existence of this kind of generalization, as hitherto it has not been taken into account, though it undoubtedly plays a significant role in the phenomena of disinhibition (see p. 175).

* Vide Pavlov, *Conditioned Reflexes*, pp. 118 et seq.

Summing up, we think the reader will agree that the physiological mechanism of similarity we have put forward fits well with the known experimental facts concerning the generalization of conditioned reflexes. The usefulness of the proposed conception will, however, appear still more clearly in the next chapter, when we shall apply it to the explanation of the phenomenon of differentiation.

CHAPTER IX

Internal inhibition. 1. Its nature and varieties

1

At the outset of our analysis of the process of internal inhibition we must note that it undoubtedly presents far greater difficulties than that of the active conditioned reflexes. For although the intimate mechanism of the *formation* of these reflexes has many unclarified aspects, the mechanism of their *course* can be regarded, within certain limits, as intelligible, because it largely coincides with the mechanism of inborn reflexes But so far as inhibitory conditioned reflexes are concerned, hitherto it has been impossible to determine what phenomena in the field of the lower nervous activity they correspond to, and, consequently, we are not in possession of a ready-made prototype for them. So a model of such reflexes has to be built up from the beginning, drawing on the known experimental facts on the one hand and the laws of the functioning of the nervous system on the other, and taking care to ensure that all the known facts fit the model. For it must be remembered that a false model, like a false prophet, can lead one badly astray in regard to both the interpretation of existing facts and the formulating of new problems.

At first glance it would seem simplest to explain the mechanism of the disappearance of non-reinforced conditioned reflexes by the gradual elimination or atrophy of the synaptic contacts formed between the conditioned and the unconditioned centres. However, there are such serious arguments against this view that it is impossible to accept it.

To begin with, the fading of intercentral connexions as the converse process to their formation would connote the return of the conditioned stimulus to its original state before conditioning. But, as we know, that is by no means the case, for the non-reinforced conditioned stimulus acquires certain new properties, which are not in the least characteristic to stimuli which have not been conditioned at all.

Secondly, there is clear evidence that a gradual fading of intercentral connexions, i.e. their retrogressive development, occurs when a conditioned stimulus, not too firmly established, is not applied at all for some time. Now an applied but non-reinforced conditioned stimulus has fundamentally different properties from a stimulus which has been conditioned, but thereafter has not been applied. Briefly speaking, the difference consists in the circumstance that while a stimulus long left unapplied simply becomes a weaker (less elaborated) conditioned stimulus, a non-reinforced stimulus becomes an elicitor of an inhibitory conditioned reflex. Speaking in psychological terms, if the conditioned stimulus is not applied the animal *forgets* that this stimulus has been conditioned, whereas if the conditioned stimulus is non-reinforced the animal *learns* that the reinforcement which previously had been presented with this stimulus is now omitted. So a theory which did not take account of the difference between two such distinct phenomena would be inadequate.

Thirdly and finally, the application of the conditioned stimulus without reinforcement is a certain *new* combination of stimuli reaching the cerebral cortex, and, as such, it must lead to the formation of a new plastic change in it. There is much evidence, both physiological and psychological, that new plastic changes by no means cause the elimination of old changes, but in some way are superimposed on them. Concretely speaking, so far as the inhibitory stimuli are concerned, it can always be shown without difficulty that they *were* previously active conditioned stimuli, and that inhibition has by no means destroyed the excitatory connexions between the conditioned and the unconditioned centre. The best evidence of this is the fact of the disinhibition of inhibitory reflexes caused either by the application of extraneous stimuli or by an increase in the excitability of the cerebral cortex. So the concept of internal inhibition as the fading of intercentral connexions is in obvious contradiction to the existing facts.

The acceptance of superimposition of new plastic changes upon the old as one of the fundamental principles of plasticity not only makes it possible to reject the conception of inhibition

formulated above, but also provides a key to the formulation of a new conception, which seems to fit the existing state of things better. Namely, we assume that non-reinforcement of a conditioned stimulus by an unconditioned stimulus causes the formation and multiplication of *new synaptic connexions of an inhibitory character side by side with old ones.* It follows that, when activated, the centre of a conditioned inhibitory stimulus sends both excitatory and inhibitory impulses to the centre of the reinforcing stimulus, and the magnitude of the conditioned reaction depends on the relative density of both the showers of impulses falling on the unconditioned centre.

It is easy to see that although the conception just put forward has still to be verified against experimental facts, it is in complete harmony with, and in a sense is consequential upon, the evidence of general physiology of the central nervous system. For if, as we assumed in Chapter IV, there are both excitatory and inhibitory synapses in the nervous system, and the state of inhibition is passed to the nerve cell by the intermediary of these latter synapses, and if, as we showed in Chapter V, the formation of an excitatory conditioned reflex depends on the formation and increase of excitatory synapses between the conditioned and the unconditioned centre, there can be no doubt that inhibitory synapses also can be formed between the cortical cells, and that they are the cause of the formation of inhibitory conditioned reflexes. The great body of experimental evidence collected by the Pavlov school proves that such inhibitory reflexes do in fact arise if the conditioned stimulus is non-reinforced by the unconditioned stimulus, i e in the phenomena covered by the term internal inhibition

So the assumption that inhibitory synaptic connexions arise between corresponding centres in an inhibitory conditioned reflex is fundamentally on the same plane as the assumption that excitatory connexions arise in excitatory conditioned reflexes, and, like the latter, the former assumption can be tested (as we shall see) by reference to experimental data. But let us try to see whether we can explain, at least to some extent, *why* the non-reinforcement of a conditioned stimulus leads to the formation of inhibitory connexions between the conditioned and

the unconditioned centre, especially in view of the fact that if an indifferent stimulus is applied in the same way (i.e. without any reinforcement) no connexions of this kind are formed.

As was said in Chapter II, investigators in Pavlov's laboratories (Kreps, Pavlova, Pietrova, Vinogradov) showed that if an indifferent and an unconditioned stimulus are applied in reverse overlapping sequence (the unconditioned stimulus being pre-current) then not only does a positive conditioned reflex fail to be established, but the indifferent stimulus is transformed into a strong inhibitory stimulus (see p. 19). Moreover, if a conditioned stimulus, relatively fresh and not firmly established, is repeatedly 'overlapped' in this way by an unconditioned stimulus, it soon loses its active conditioned properties and is transformed into an inhibitory stimulus.*

Pavlov himself was extremely puzzled by these facts and looked for their appropriate explanation.† Finally, he concluded that evidently if a given cortical point is repeatedly stimulated when subjected to external inhibition, it gains negative excitability and becomes permanently inhibitory

We have found that Pavlov's explanation is inaccurate and that the facts under consideration must be interpreted in quite another way.‡ We have shown by experiments that if an indifferent stimulus is applied after the inception of an unconditioned alimentary stimulus but is reinforced by an additional portion of food which protracts the act of eating, then a positive conditioned reflex is formed to this stimulus, this reflex being stable and of considerable intensity (fig. 9*c*). But if the additional portion is withheld, and the conditioned stimulus applied during the act of eating signalizes the termination of this act, then the conditioned reflex very quickly undergoes extinction, i.e. is inhibited (fig. 9*d*). From this we drew the conclusion that in the above-described experiments of Pavlov's collaborators the reverse overlapping sequence of the stimuli led to the formation of an inhibitory conditioned reflex, not

* Podkopayev, N A, *Trudy Lab. Pavlova*, 1928, vol II/2, p 81; Right (Rait), R. G., ibid p. 87, Soloveychik, D. I, ibid p. 95

† See Pavlov, *Lectures*, vol. I, pp 381–3.

‡ Konorski, J, Lubinska, L. and Miller, S., *Acta Biol. Exp* 1936, vol. x, p. 297.

because the conditioned stimulus acted against the background of the inductively inhibited cerebral cortex, but because it preceded and signalized the discontinuance of the unconditioned stimulus Generally speaking, our experiments, supported by the evidence of the above-cited authors, demonstrated that, if an indifferent stimulus precedes the *termination* of an unconditioned

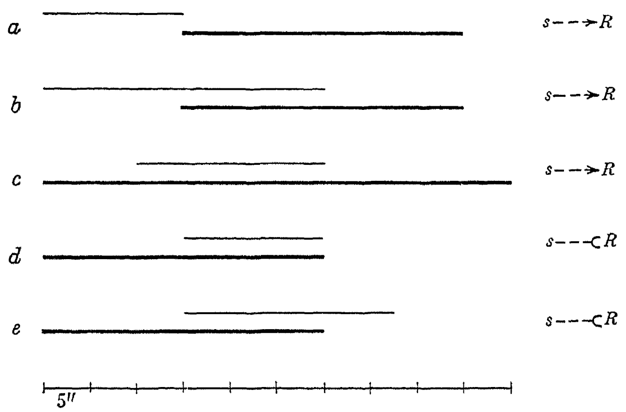


Fig 9. The effect of the stimuli sequence upon the formation of the conditioned reflex. Thin lines denote the duration of the 'indifferent' stimulus, thick lines denote the duration of the reinforcing stimulus (*a*) The indifferent stimulus precedes the reinforcing stimulus: as the result excitatory conditioned reflex is established (*b*) The two stimuli are applied in overlapping sequence. this is the optimal sequence for elaboration of the excitatory conditioned reflex. (*c*) The indifferent stimulus is applied during the operation of the reinforcing stimulus which is protracted the excitatory conditioned reflex is established, its magnitude being less than in (*b*) (*d*) The indifferent stimulus precedes the termination of the reinforcing stimulus the inhibitory conditioned reflex is established (*e*) Two stimuli are applied in reverse overlapping sequence: the strong inhibitory conditioned reflex is established *s*-->*R*, excitatory conditioned reflex; *s*--<*R*, inhibitory conditioned reflex Time (5 sec.)

stimulus, an inhibitory conditioned reflex is established in just the same way as an excitatory conditioned reflex is formed when a stimulus to be conditioned precedes the beginning of an unconditioned stimulus.

The removal of an unconditioned stimulus connotes a more or less abrupt *fall* of excitation of the corresponding centre,

i.e. a phenomenon exactly converse to that which occurs when by applying the unconditioned stimulus the excitation in this centre is suddenly raised. In Chapter VI (§ 4) we demonstrated that when the excitation of a given cortical centre is synchronous with the rise of excitation in another centre, conditioned excitatory connexions are formed from the first of these centres to the latter. Now we find that, conversely, the excitation of a given cortical centre synchronized with the fall of excitation in another centre results in the establishment of inhibitory connexions between these two centres.

It is easy to grasp the similarity between this method of elaboration of an inhibitory conditioned reflex, and those methods used in everyday practice in conditioned reflex experiments. Let us assume that a conditioned alimentary stimulus is applied without reinforcement. This stimulus produces active excitation of the alimentary centre, of which the visible sign is the secretion of saliva, and motor alimentary reaction. But food is not presented, and therefore excitation of the alimentary centre is abruptly falling. Thus the conditioned stimulus, which so far has been a signal of the rise of excitation in the alimentary centre, now becomes the signal of its fall This causes inhibitory connexions to form between the centre of the conditioned stimulus and the alimentary centre, on the same basis as in the experiments just adduced The difference between the situation arising in the foregoing experiment and that in ordinary cases of inhibition is solely that, whereas in the first case the excitation of the unconditioned centre has been produced by the unconditioned stimulus, and its fall by the discontinuance of this stimulus, in ordinary cases of internal inhibition the excitation is produced by a conditioned stimulus, and its fall is due to the absence of reinforcement.

From this it follows that the primary and direct method of forming a stable and strong inhibitory reflex consists in reinforcing an indifferent stimulus by discontinuing the unconditioned stimulus, whereas the methods generally used in laboratory practice are rather indirect. Indeed, the inhibitory reflex obtained by the reverse sequence of stimuli is the direct converse of the excitatory reflex formed by a normal sequence of stimuli (cf.

fig. 9*b* and *e*), while the inhibitory reflexes dealt with in Pavlov's school correspond rather to the conditioned reflexes of the second order. Unfortunately, the method of elaboration of inhibitory conditioned reflexes just described has not been applied so far in experimental practice, and we have willy-nilly to utilize only the experimental material at present at our disposal.

To end these general remarks on the mechanism of inhibitory conditioned reflexes, we would like to point out that, as follows from our conception, cells of the conditioned centre and those of the unconditioned centre are linked together both by excitatory and by inhibitory connexions. If one accepts (as for the sake of prudence we did on p. 71) that each axon can provide terminals of only one kind, it has to be assumed that the connexions between the two centres can never be direct, but possess at least one intermediary relay. As we have pointed out above (Chapter VI, § 1), such an assumption is entirely justified

2

Taking the conception just advanced as a basis, we turn now to examination of the various kinds of internal inhibition. We begin with the simplest kind of inhibition, namely, experimental extinction.

As we have said, the non-reinforcement of a conditioned by an unconditioned stimulus leads to the formation of inhibitory connexions between the centres of the two stimuli, and with repetition of the non-reinforced conditioned stimulus these connexions grow more and more numerous In consequence, every time the conditioned centre is excited it sends more and more inhibiting impulses to the unconditioned centre, and these more and more effectively oppose the simultaneously sent exciting impulses. The conditioned reflex diminishes more and more, and at last completely disappears. The stronger the conditioned stimulus, i.e. the more excitatory connexions exist between the two centres, the more inhibitory connexions must be formed in order to subject the conditioned reflex to complete inhibition. So the total extinction of such a reflex follows after a larger number of trials than that of a conditioned reflex to a weak stimulus. Similarly, a firmly established conditioned reflex

becomes extinct more slowly than a fresh one, since its reflex arc also possesses more excitatory connexions (vide p. 101).

If an acutely extinguished conditioned stimulus is not repeated for a period of time the fresh inhibitory connexions fade, just as do fresh excitatory connexions when a positive conditioned reflex is not trained. Therefore, if a firmly established conditioned reflex is subjected to experimental extinction and then the application of the extinguished stimulus is suspended for several hours, the excitatory conditioned reflex is generally thereafter fully restored. This is explained by the very poor stability of the fresh inhibitory connexions as compared with the much greater stability of the old and firmly established excitatory connexions (cf. Chapter v, § 2). On the contrary, if an old and firmly established inhibitory reflex is acutely transformed into an excitatory conditioned reflex the firmly established inhibitory connexions will prove to be stable, while new excitatory connexions will be liable to atrophy As a result, after a lapse of time, the new excitatory conditioned reflex will disappear and the inhibitory reflex will be fully restored (see below).

These facts not only constitute an illustration of the evidence showing that the stability of synaptic connexions (both excitatory and inhibitory) depends upon the training of the corresponding reflex, but also yield a proof that old excitatory connexions are not annihilated by the new inhibitory connexions, and vice versa, but new connexions are formed side by side with the old ones.

As we said in Chapter iv, the effect of inhibitory impulses on the nerve cell can be considered as consisting in a rise in the threshold of its excitability. It follows that the cells of an unconditioned centre affected by inhibitory impulses temporarily change their characteristic so that the curve *ABC* in fig 4 (p. 73) is shifted to the right, and this the more, the greater the number of inhibitory connexions formed between the two centres The shape of the curve *ABC* of course remains the same, since the discharge capacity of the cells themselves does not undergo any change.

It is worth pointing out here that there was a similar shift of the curve *ABC* to the right in the case of lowered excitability of the unconditioned centre (cf. fig. 4, p. 73 and fig. 6, p. 104). The

difference between the two phenomena consists in the fact that, whereas in the latter case the change in characteristic is of a more permanent character, i.e. it lasts as long as there is a state of decreased excitability, in the case of inhibition the change in the threshold is acute, and lasts only as long as the inhibiting impulses are falling on the nerve cells

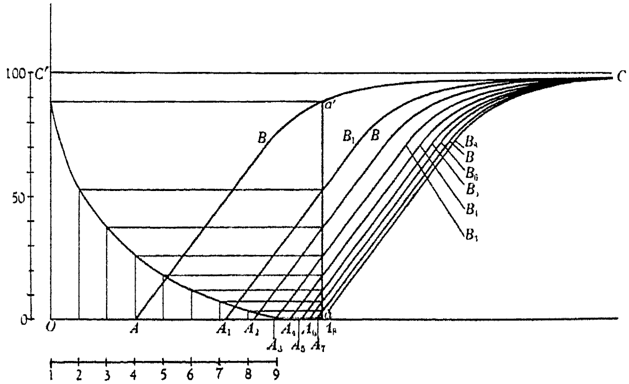


Fig 10 The process of extinction of the conditioned reflex Abscissa (1) The number of applications of conditioned stimulus without reinforcement (lower line) (2) The quantity of excitatory impulses falling on the unconditioned centre in a unit of time Ordinate The size of the conditioned reflex response The stimulus used for extinction, acting against the background of 'normal' characteristic curve of alimentary centre *ABC*, evokes the response *aa'*. The curve of extinction is constructed according to the actual experimental data From the diminution of the reflex response at each extinction trial the shift of the characteristic curves to the right can be deduced ($A_1B_1C_1$, $A_2B_2C_2$, etc) provided that each application of the stimulus produces a shower of excitatory impulses of the same density, hence the increase of the density of inhibitory impulses falling on the centre at each trial (AA_1, A_1A_2, etc) can be calculated, it corresponds to the increase of inhibitory connexions at each trial As is seen from the diagram, the increase of inhibitory connexions decelerates as the process of extinction proceeds Further explanation in text

The shape of the extinction curve of the conditioned reflex is of great theoretical importance, since its slope at any given point represents the rate of the formation of the inhibitory connexions between the conditioned and the unconditioned centre. As is well known, the typical extinction curve of a conditioned reflex is concave, i.e. the size of the reflex response at

first diminishes very rapidly, but then more and more slowly. This fact is very significant, since it proves that the rate of the formation of inhibitory connexions diminishes as the number of connexions increases.

The above-stated conclusion can be presented graphically as follows (fig. 10). The size of the conditioned reflex is plotted against the number of applications of the non-reinforced conditioned stimulus (for the curve of extinction) and against the density of the excitatory impulses falling on the cells of the unconditioned centre (for the curves ABC, $A_1B_1C_1$, $A_2B_2C_2$, etc.). In fig. 10 the curve of extinction of the conditioned reflex is taken from one of our experiments.* The initial size of the reflex response was near to the top value. With the increase of inhibitory connexions between the conditioned and the unconditioned centre there was a gradual diminution in the size of the reflex response, and from the extent of this diminution it is possible to infer how far the curve of the characteristic is shifted. The distances between the various curves, i e. the intercepts AA_1, A_1A_2, A_2A_3, etc., measure the increase of inhibitory connexions at each trial. As can be seen from the diagram, this increase proceeds more and more slowly, in line with the process of extinction, i e. with the augmentation of the number of inhibitory connexions formed.†

* Konorski, J , *Rev. Physiol Mouvt* , 1939, vol. ix, p. 191, fig 6.

† However, it must be pointed out that there is not a complete analogy between the decelerating increase of excitatory connexions in the elaboration of an excitatory conditioned reflex, and the decelerating increase of inhibitory connexions in the extinction of a conditioned reflex In the first case the conditioned stimulus is accompanied by the rise of active excitation in the unconditioned centre, due to the presentation of food This rise is exactly the same at each trial, provided the alimentary excitability is unchanged But in extinction the active excitation of the alimentary centre evoked by the conditioned stimulus diminishes with each trial, because of the inhibitory connexions which are being formed in larger and larger quantities. So the deceleration in the increase of these connexions probably depends not only on their approximation to a state of 'saturation', but also on the circumstance that with each successive trial the active excitation of the alimentary centre is weaker, and therefore, when food does not follow its fall is smaller An experiment completely analogous to those concerned with the formation of an excitatory conditioned reflex would be made if the stimulus (whether conditioned or indifferent) were reinforced by a cessation of the act of feeding, i e by a fall of excitation, caused by the

However, when analysing the curve of extinction it must be remembered that its shape depends on a number of factors which may deform it more or less. Thus, if the intervals between the applications of the extinguished stimulus are too long (e g if extinction is more chronic) it is necessary to take into account the regression of the fresh inhibitory reflex in the intervals between successive trials, a circumstance which greatly complicates the course of the curve. On the other hand, if the extinction of the conditioned reflex is acute, and if we have to do with an alimentary reflex, then the course of the extinction may be disturbed (especially in certain animals) by the inertia of inhibition in the alimentary centre, a phenomenon which we shall discuss in detail in the next chapter.

Extinction (whether acute or chronic) to zero of the conditioned reflex by no means indicates that the inhibition of the unconditioned centre has reached the peak point and will not grow any further. First, with further repetitions of the non-reinforced stimulus the inhibitory reflex grows more stable in the sense that it becomes more and more insusceptible to a break in its elicitations, which shows that the inhibitory connexions are strengthened. Secondly, inhibitory connexions can continue to increase (if their quantity has not reached saturation point) exactly like excitatory connexions after the conditioned reflex has reached its top This is shown chiefly by the fact that the more deeply a conditioned reflex is extinguished (i e. the more the stimulus has been repeated without reinforcement after zero reaction was achieved), the more reinforced trials are required to restore the conditioned reflex.* Apart from this, numerous proofs of the fact that the increase of inhibitory connections continues after the conditioned reaction has reached zero will be given in the next chapter.

The important question arises, what happens to the *excitatory*

removal of the unconditioned stimulus It is clear that this fall is always approximately the same, just like the rise of excitation following the presentation of food As there is no experimental evidence covering this case, we are not in a position to conduct a comparative analysis of these two cases of inhibition.

* Vide experiments by Kasherininova, N. A , Diss , Petersburg, 1908, p 97

connexions as the non-reinforced conditioned stimulus is repeated?

So far as acute extinction is concerned, there is hardly any doubt that these connexions do not suffer any change during this process. The best proof of this is the above cited fact of the complete restoration of the conditioned reflex after a longer or shorter lapse of time. The idea that excitatory connexions could be destroyed during the extinction and afterward could spontaneously 'regenerate' seems both arbitrary and unnecessary.

However, the position may be different in the case of chronic extinction. For in this case the conditioned stimulus is not reinforced for a period of many months, and even years. It is doubtful whether in these conditions the excitatory connexions can be maintained, it is rather to be assumed that they undergo atrophy. Maybe this atrophy is precisely the same as if the stimulus were not applied at all, and so it would depend on the degree to which the conditioned reflex to this stimulus had been stabilized; it is also possible that the application of a conditioned stimulus without reinforcement in some way accelerates or retards this atrophy. In any case, if its existence is assumed, it must in turn be accepted that inhibitory connexions also atrophy, since they are 'nourished' by the fall of excitation in the unconditioned centre, and so cannot be maintained if this excitation ceases to operate.

So we arrive at the conclusion that a conditioned stimulus, repeated over a *very* long period of time (i.e. months and years) without reinforcement, slowly becomes an indifferent stimulus, i.e. its centre loses all actual connexions with the corresponding unconditioned centre. Intuitively, this deduction seems to be very probable, but there is no evidence whatever in substantiation of it, since the process of chronic extinction has hardly been investigated at all. Yet it would not be difficult to test this deduction Indeed, if it were established that the stimulus now under discussion no longer caused any inhibitory after-effect, did not give positive induction and could not be disinhibited in any way, we would then have no evidence whatever for regarding it as an inhibitory stimulus

On the basis of the foregoing remarks we must conclude that the process which Pavlov called 'extinction with reinforcement' has nothing in common with the phenomena of extinction in the strict sense of this term. It is difficult to determine the mechanism of this phenomenon, owing to the lack of the appropriate control experiments. Taking into account the fact that 'natural' conditioned stimuli (such as scent, or the sight of food) do not succumb to 'extinction with reinforcement', despite the fact that nature presents them throughout an animal's life, we must conclude that it is associated with some aspect of experimental technique. So far as 'extinction with reinforcement' with an acute course is concerned, i e. one which arises in a single experimental session as the result of the multi-repetition of the same conditioned stimulus,* this is probably simply a case of the 'fatigability' of the corresponding intercellular connexions, plus the animal's satiation (for the experiments were conducted exclusively with alimentary reinforcement). In the case of chronic 'extinction with reinforcement'† when, after the stimulus has been applied for many months and years, its effect begins gradually to diminish and may finally disappear altogether, presumably we have to do with the formation of a delayed reflex. Certainly, if the reinforcing stimulus is continually postponed by 15–30 seconds from the beginning of the conditioned stimulus, the animal finally learns that the food does not follow immediately after the application of the conditioned stimulus, but only some time later. In consequence, a strong and lasting inhibitory reflex is formed to the initial phase of the conditioned stimulus.

But while 'extinction with reinforcement' can never be considered as a 'true' extinction, the position is quite otherwise with regard to a diminution of the conditioned reflex as the result of a *weakening* of the unconditioned stimulus.

In Chapter VI, when discussing the properties of conditioned reflexes, we drew attention to the circumstance that the magnitude of a conditioned reflex depends, *inter alia*, on the strength

* Stroganov, V. V , *Trudy Lab. Pavlova*, 1929, vol III/2–3, p 103. Soloveychyk, D. I , ibid 1940, vol IX, p 277.

† Pavlov, *Conditioned Reflexes*, chap XIV

of the reinforcing unconditioned stimulus We explained this fact by assuming that a stronger unconditioned stimulus produces excitation in a larger cellular field, and in consequence the cells of the conditioned centre form connexions with a greater number of cells of the unconditioned centre It follows that a lessening of the conditioned reflex caused by a weakening of the reinforcing unconditioned stimulus (e.g. by a diminution in the portion of food presented) should occur by way of a partial extinction of this reflex, since a part of the cells of the unconditioned centre excited by a conditioned stimulus is not successively excited by the reinforcing stimulus, with the result that these cells should receive inhibitory connexions from the conditioned centre. In fact, as Kleschchov's thorough and detailed experiments showed,* a lessening of conditioned reflexes in consequence of a diminution of the portion of food presented with the alimentary conditioned stimulus occurs as the result of the process of internal inhibition. While from the viewpoint of the Pavlov theory this result is rather unexpected, from our viewpoint it is quite understandable and could easily be foreseen.

In conclusion to this section we should like to remark that the mechanism of extinction of the orientation reflex (*vide* Chapter v, § 1) has to be thought of as analogous to that of extinction of the conditioned reflex. In other words, we assume that the gradual disappearance of the orientation reflex with the repetition of the corresponding stimulus is due to the formation of inhibitory connexions between certain intervening centres in just the same way as they are formed in the extinction of the conditioned reflex. This would explain the striking similarity of the properties of these two processes. for instance, when the application of the extinct stimulus is suspended for some time, the orientation reflex tends to recover spontaneously in just the same way as does the extinguished conditioned reflex; the extinguished orientation reflex can be disinhibited by another extraneous stimulus, etc. The only difference between the two processes is that whereas the excitatory conditioned reflex and the superimposed inhibitory conditioned reflex are both acquired and are dependent

* Kleshchov, S. V , *Trudy Lab Pavlova*, 1936, vol vi, no 2.

mainly on the cerebral cortex, the orientation reflex itself is inborn and of subcortical origin, unlike its inhibition which, being formed on the basis of the animal's individual experience, originates chiefly from the cerebral cortex.

3

Before we proceed to consideration of the other kinds of internal inhibition we have to discuss a problem of great theoretical importance, namely, that of the mechanism involved in re-establishing an excitatory conditioned reflex to an inhibitory stimulus, this latter being again applied with reinforcement. Unfortunately, we are not in a position to provide a solution to this problem, because of the almost complete lack of appropriate experimental evidence.

Experiments with the re-establishment of conditioned reflexes have been conducted only in relation to acute extinction and differentiation. The first of these cases is unsuitable for analysis, since the inhibitory connexions are so fresh that they fade spontaneously after a lapse of time and so the conditioned reflex recovers even without retraining. In the second case inhibitory connexions are certainly adequately fixed, but the fact that here we have to do with a differential inhibition greatly complicates the experimental situation (see below). On the other hand, no experiments have been performed with the re-establishment of chronically extinguished reflexes, and this is precisely the form of experiment necessary to elucidation of the problem we have posed.

The experimental evidence we have at our disposal indicates that the reinforcement of even a firmly established inhibitory reflex leads, generally speaking, to the re-establishment of a positive conditioned reflex to it (if cases of pathological stability of the inhibitory reflex are left out of account), and that this reflex may achieve the normal value it had before inhibition. The rate of formation of such a reflex varies greatly, and is dependent on various factors; but the fact is worthy of note that application of the stimulus with reinforcement several times successively leads to an almost immediate re-establishment of an active conditioned reflex in a single experimental session. Such

a reflex, however, is very unstable, and may subside completely the very next day.

Let us assume that a conditioned reflex has been elaborated to a stimulus, and that in the course of further training this reflex has been adequately stabilized. Then, by giving this stimulus without reinforcement day after day, the reflex undergoes chronic extinction, and the inhibitory reflex thus formed is also trained for a period. Then the stimulus is again reinforced by the unconditioned stimulus until the active-conditioned reflex is re-established. The question arises, what are the changes in the intercentral connexions that cause this secondary formation of the conditioned reflex?

The simplest of the possible explanations of this phenomenon would be as follows. During the chronic extinction of a conditioned reflex which, be it remembered, has lasted for a long period, at least certain of the excitatory connexions might be atrophied, and with a renewed reinforcement of the stimulus these would again 'regenerate'. On the other hand, reinforcement of the inhibitory stimulus may lead to a gradual disappearance of the inhibitory connexions, just as happens, according to our assumption, in the case of excitatory connexions as the result of non-reinforcement of the conditioned stimulus. Thus the excitatory connexions finally gain the upper hand over the inhibitory connexions, and so the active conditioned reflex is restored.

However, it must be pointed out that though in all probability this explanation is correct to a certain extent, it cannot be completely satisfying. First and foremost it is hardly believable that well-fixed excitatory connexions could undergo any considerable atrophy in the course of the few months required for the formation of a stable inhibitory reflex. Secondly, as we have already pointed out, even a very firmly established inhibitory reflex can be transformed into an active reflex in the course of a single experiment. Truly, this occurs with differential inhibitors, but there is no reason to doubt that it also may occur in the case of chronic extinction. Now in a single experiment inhibitory connexions cannot undergo *any* atrophy, a fact of which there is direct proof in the circumstance that the inhibitory reflex is spontaneously restored the very next day.

The fact just cited is deserving of considerable attention. It is known that if the conditioned reflex has not been repeated for a long time, so that it has undergone a certain regression, only a few trials are required to restore it to its previous state, and then the return to the standard is *permanent* at once. But in the present case the recovery of the active reflex is transient, from which we must deduce that the excitatory connexions formed are *fresh*. However, the question arises, how can fresh excitatory connexions be formed if the original conditioned reflex was firmly established, and the corresponding intercentral connexions which were then created were undoubtedly in a state of complete saturation?

The conclusion that would seem to result from this analysis is that the quantity of excitatory synapses between the two centres, which has reached saturation point after the first formation of the conditioned reflex, becomes insufficient for saturation when inhibitory connexions are formed between these centres. In other words, the inhibitory connexions would seem to increase the 'capacity' of the surfaces of the receiving cells to establish excitatory synapses, and so after the formation of inhibitory connexions between the centres 'room is made' for new excitatory connexions.

This conclusion, which may seem strange at first sight, has a number of arguments in its support. For, as follows from the considerations adduced in Chapter v, the main force causing the formation of excitatory connexions between two concurrently activated coupled centres is the lack, or the inadequacy of these connexions, so that the impulses arriving from the emitting centre are unable to activate, or inadequately activate, the receiving centre. As, owing to the formation of new connexions, the 'transmissibility' between the two centres increases, the formation of new synapses proceeds at a decelerating rate, and finally ceases altogether. Now, the establishment of inhibitory connexions between these centres in a certain sense restores the former relation between them. Namely, excitatory impulses coming from the emitting centre are again unable to produce active excitation in the cells of the receiving centre, in this case because of the hindering effect of the inhibitory impulses. So it

may be that in such conditions, when concurrent excitation of both centres is renewed, excitatory connexions are formed anew, until the transmissibility between these centres again becomes optimal, i.e. until the excitatory connexions reach a new state of saturation.

So then, while previously we treated the increment of excitatory connexions as an inverse function of already formed excitatory connexions, now it would be necessary to modify this view somewhat, by saying that their increment is an inverse function of the transmissibility between the two centres, i.e of the ease with which the activation of the emitting centre is able to produce active excitation in the receiving centre It is easy to see that such a formulation embraces both the formation of excitatory conditioned reflexes to 'indifferent' stimuli, and the formation of such reflexes to inhibitory stimuli.

Ending this section of our argument, we think it worth while recalling yet again that it has no direct support in experimental facts, and that our deductions have been extrapolated from experiments which only indirectly testify in their favour. And so the problem we have been discussing calls for detailed experimental investigation, and only the appropriate facts will be able to inform us whether the state of saturation of the excitatory synapses between two coupled centres really does depend on the inhibitory connexions existing between them, and vice versa, or whether this supposition is incorrect, and the re-establishment of an active conditioned reflex to an inhibitory stimulus depends solely on the regeneration of faded excitatory connexions and the fading of the inhibitory connexions

4

Now we turn to a discussion of the next type of internal inhibition, namely, differential inhibition. It arises when a new stimulus similar to the conditioned one is introduced into experiments and then applied without reinforcement. As, because of generalization, this stimulus also is a conditioned stimulus, its non-reinforcement leads to the formation of an inhibitory reflex to it.

In our preceding chapter we gave reasons for the view that

generalization is caused by a partial overlapping of the centre of the original conditioned stimulus with the centres of the secondary stimuli, and that, the more they overlap, the stronger is the degree of generalization. Let us attempt on the basis of this conception to explain the mechanism of differentiation.

Let us assume that the stimulus s_1 is the primary conditioned stimulus, reinforced by the unconditioned stimulus S, and that the stimulus s_2 similar to it has been differentiated. In the centre of the stimulus s_1 we can distinguish the subcentre a, belonging only to the centre s_1 and not belonging to the centre s_2, and the subcentre b, common to both centres. Similarly, the centre s_2 can be divided into subcentre b and subcentre c, the latter belonging only to the centre s_2 Now let us consider what connexions will arise between each of these three subcentres and the unconditioned centre during the process of differentiation and after it is completed

Subcentre a, appertaining only to the centre s_1, at each excitation is reinforced by the excitation of the unconditioned centre both in the period before differentiation, and during the formation of differentiation. So the synaptic connexions of this subcentre with the unconditioned centre are exclusively of an excitatory character.

The situation of subcentre b is more complicated. So long as only the conditioned stimulus s_1 is given, this subcentre possesses exclusively excitatory connexions with the centre S, but then, when the stimulus s_2 is introduced into the experiments, being given without reinforcement, this subcentre forms inhibitory connexions with the unconditioned centre. Now, however, the position is completely different from that arising in the case of extinction of a conditioned reflex In that case the excitation of a conditioned centre occurred without reinforcement throughout the training (i.e. during the period of formation and maintenance of the inhibitory reflex), while in the case of differentiation the excitation of subcentre b *is* at one time associated with the direct excitation of the unconditioned centre (when this subcentre shares in the excitation of the centre s_1), and at another time is *not* associated with this excitation (when it shares in the excitation of the centre s_2). So although between the subcentre b

and the centre S inhibitory connexions are formed, yet the excitatory connexions continue to be trained and cultivated.

Obviously here we are confronted with the problem we have already discussed in detail in the preceding section. If we accept the hypothesis which we there defended and justified, that the formation of inhibitory connexions between two centres creates conditions for the emergence of new excitatory connexions between them, then the position of the subcentre b will be as follows: each time it is excited without reinforcement will lead to the formation of inhibitory connexions between it and the unconditioned centre, but when it is excited with reinforcement the existence of newly established inhibitory connexions will render possible the formation of new excitatory connexions. The general 'balance' of the excitatory and inhibitory connexions will therefore depend on the comparative frequency with which the stimuli s_1 and s_2 are applied, and will be approximately stable. Therefore the difference between the position of the subcentre b and that of the conditioned centre in extinction is clear. In extinction only inhibitory connexions are formed against the background of an invariable number of excitatory connexions, but here the excitatory and inhibitory connexions are formed parallel, since one group 'makes room' for the other, and vice versa.

The conclusion we have just reached on the basis of our previous hypothesis seems to be in complete harmony with the generally known experimental data. Namely, it is known that in the first stage of differentiation a conditioned reflex to the stimulus s_1 (i.e. the positive conditioned stimulus) diminishes, which in our opinion is an expression of the rapid formation of inhibitory connexions between subcentre b and the unconditioned centre. However, in the following stage the reflex to stimulus s_1 is restored, which has to be explained as compensation by the newly arising excitatory connexions. As we said in Chapters II and III (pp. 20 and 41), it often happens that a conditioned reflex to the stimulus s_1 becomes greater after differentiation than it was before. Obviously it is difficult to explain this fact without special experiments, but we think we are entitled to express the supposition that it takes place

because of some hyper-compensation on the part of the newly established excitatory connexions. In any case this increase of the conditioned reflex to the stimulus s_1 indicates that some kind of 're-organization' has taken place between the centre of this stimulus and the unconditioned centre, this 'reorganization' consisting, perhaps, in the formation of new excitatory and inhibitory connexions between subcentre b and centre S.

However, we must remember that our hypothesis has not so far been directly confirmed, and that the difference between the position of subcentre b in differentiation and the conditioned centre in extinction of the conditioned reflex may perhaps be explained in a totally different manner: in the extinction of the conditioned reflex the excitatory connexions are not trained, and in consequence they partly atrophy, whereas during differentiation the excitatory connexions between the subcentre b and the centre S are constantly trained, and so are maintained in a state of saturation.

As for the subcentre c, belonging to the centre s_2 and not belonging to the centre s_1, its excitation never coincides with the excitation of the unconditioned centre by the unconditioned stimulus. On the other hand, it is constantly excited together with subcentre b, producing an excitation of the unconditioned centre by way of conditioned connexions, and so its excitation coincides with the fall of excitation in this centre Consequently only inhibitory connexions can be formed between the subcentre c and the unconditioned centre, if one leaves out of account those few excitatory connexions which could arise through the formation of a conditioned reflex of the second order (cf. p 107). From the foregoing it follows that the main burden of the differentiation rests precisely on this subcentre c, since it must provide the unconditioned centre with an adequate quantity of inhibitory connexions to balance the great quantity of excitatory connexions formed between subcentre b and the centre S.

Now let us try on the basis of the foregoing analysis to explain the main properties of differentiation. To begin with we shall consider what is the difference between so-called 'fine' and 'coarse' differentiation.

Fine differentiation arises when the stimulus s_2 is very similar

to the stimulus s_1, i.e. when their centres overlap to a comparatively large extent. It follows that the excitation of the centre s_2 elicits a strong excitation in the unconditioned centre, and consequently, the stimulus s_2, being non-reinforced, gives rise to a strong inhibitory reflex. We have evidence that this actually does happen, as is shown, for instance, by the prolonged and strong inhibitory after-effect following a differentiated stimulus when the inhibitory reflex to it has been firmly established. However, fine differentiation is also 'difficult', i e. it is elaborated slowly, since its achievement demands the formation of a large number of inhibitory connexions, to balance the abundant excitatory connexions existing between the subcentre b and the centre S. For the same reason also it is unstable, is easily 'spoilt', or suffers disinhibition. This will be discussed at length in the next chapter. The existence between the centre s_2 and the centre S of a large number of both excitatory and inhibitory connexions has the effect that the inhibitory reflex to the stimulus s_2 possesses peculiar 'tension', as if a severe struggle were occurring between the processes of excitation and inhibition.

If differentiation is *too* fine, i.e if the part c of the centre s_2 cannot form a sufficient quantity of inhibitory connexions with the unconditioned centre for them to balance the numerous excitatory connexions coming from part b, it remains constantly incomplete, requires continual training (with the danger, however, of overtraining), and easily succumbs to weakening as the result of too frequent application of the conditioned stimulus s_1. This, of course, occurs because the repetitive application of this stimulus with reinforcement results in the predominance of the excitatory connexions between the centres b and S, and in consequence the inhibitory reflex to the stimulus s_2 is disinhibited.

On the other hand, in 'coarse' differentiation the common part b of the centres s_1 and s_2 is not large, with the result that the stimulus s_2 weakly excites the unconditioned centre, and so the inhibitory reflex formed to it is comparatively weak. At the same time it is formed without difficulty, because of the small quantity of excitatory impulses being sent by subcentre b. We say of such a differentiation that it has low 'tension'.

The above-described relations could be analysed with far greater detail and precision if, instead of using simple and 'indivisible' stimuli, compound stimuli were used, e.g. if the compound stimulus s_1s_2 were differentiated from the similar compound stimulus s_2s_3. (The stimuli s_1, s_2 and s_3 must, of course, be dissimilar stimuli.) It is easy to see that the centre of the stimulus s_1 corresponds to the subcentre a, the centre s_2 to the subcentre b, the centre s_3 to the subcentre c. As it is possible to apply each of these stimuli separately and thus to excite the corresponding centres independently (which could not be done with the subcentres a, b and c), the investigator would be able to examine the properties of each of them in detail, and thus penetrate more deeply into the mechanism of differentiation. Experiments of this kind exist only in one case, namely, when the conditioned stimulus is differentiated from a compound stimulus consisting of the conditioned stimulus and an accessory agent. This case is known in Pavlovian terminology as conditioned inhibition, and it was investigated in fair detail. We shall take up its analysis in a moment.

As we have said in preceding chapters, the application of a stimulus intermediate between two differentiated stimuli results in a greater or lesser conditioned response, according to which of the stimuli the applied stimulus more closely approximates to. This is quite understandable The more the centre of such a stimulus overlaps with the centre of the conditioned stimulus, the greater is the predominance of excitatory over inhibitory connexions; the more its centre overlaps with the centre of the inhibitory stimulus, the greater is the predominance of the inhibitory connexions. So the 'balance' between the excitatory and inhibitory impulses falling on the cells of the unconditioned centre determines the magnitude of the response to the applied stimulus.

5

As we said above, conditioned inhibition is a form of differentiation exceptionally convenient for analysis, since particular components of the inhibitory stimulus can be applied, and so analysed, separately. As this form has also been investigated experimentally in detail, it can help greatly towards verification

of our conception. For whereas in our analysis of differentiation so far we have operated with wholly abstract and imperceptible parts of centres, in this case we can deal with comparatively more concrete, and in any case more operable centres of separate stimuli

Let us denote the conditioned stimulus, s_1, and the additional stimulus which with the stimulus s_1 forms the inhibitory compound, s_0.

As the stimulus s_1 *is* generally reinforced by the stimulus S (with the exception of those cases in which it is applied together with s_0), we can infer that, after the elaboration of differentiation, between its centre and the centre S excitatory connexions continue to predominate, i.e. that the inhibitory connexions arisen between them have been adequately compensated by the additional excitatory connexions. It follows that the magnitude of the conditioned reflex to the stimulus s_1 suffers no change, and in any case is not diminished. We know that this is so in fact.

On the other hand, the stimulus s_0 which is combined from time to time with the stimulus s_1 is *never* reinforced, and so exclusively inhibitory connexions must have been formed between the centre of this stimulus and the unconditioned centre; if, of course, we leave out of account those weak excitatory connexions which might be formed if this stimulus happened to be transiently a conditioned stimulus of the second order. So in the case of conditioned inhibition we deal with an interesting situation, and one very suitable for analysis, in which the excitatory and inhibitory connexions have their starting point in two different cortical centres: the centre s_1 is the starting point for almost exclusively excitatory connexions with the centre S, while the centre s_0 is almost a pure source of inhibitory connexions.

Now we shall see whether this view explains the existing experimental facts concerning this kind of inhibition.

To begin with, it is obvious that if the stimulus s_0 is much weaker than the stimulus s_1, the conditioned inhibition is formed with difficulty and often remains incomplete, since the inhibitory connexions arriving from the centre s_0 at the centre S are

inevitably weaker than the excitatory connexions between the centres s_1 and S. This fact is completely confirmed by numerous experiments.*

Secondly, if the stimulus s_0, which hitherto has been applied only in combination with the conditioned stimulus s_1, is added to another conditioned stimulus s_2, the effect of this stimulus also suffers inhibition. This is understandable, since the inhibitory impulses arriving from the centre s_0 at the unconditioned centre prevent the excitatory impulses arriving from the centre s_2 from activating it. Moreover, if the conditioned inhibitor is added to the reinforcing unconditioned stimulus, its effect also is diminished, this constituting a *direct* proof of the inhibitory effect of the inhibitor on the unconditioned centre.

Thirdly, in Leporsky's experiments† it was found that a conditioned inhibitor capable of completely inhibiting the effect of each of the conditioned stimuli separately failed to do so when they were applied jointly. This result is completely explained by reference to a balance of excitation and inhibition on the cell surfaces of the unconditioned centre.

Fourthly and finally, if the complex $s_0 s_1$ is repeated many times, and then the stimulus s_1 is applied, the reflex to it proves to be strongly and protractedly inhibited.‡ This is explained by the fact that successive repetition of the stimulus s_1 without reinforcement leads to the formation of fresh inhibitory connexions between the centre s_1 and the centre S. These connexions did not reveal themselves until this stimulus was applied together with the stimulus s_0 (since the effect of the reflex was zero in any case), but became manifest when it was applied separately. As these connexions were fresh, after a certain time they spontaneously disappeared, and in consequence the conditioned reflex to the stimulus s_1 was restored.

On the other hand, on the basis of our conception we must categorically oppose Pavlov's statement that the conditioned inhibitor inhibits not only homogeneous but also heterogeneous

* Vide Fursikov, D. S., *Trudy Lab. Pavlova*, 1924, vol. I/1, p. 3.

† Leporsky, N. I., Diss., Petersburg, 1911 (Pavlov, *Conditioned Reflexes*, p. 79).

‡ Diegtarova, V. A., Diss., Petersburg, 1914 (Pavlov, *Conditioned Reflexes*, p. 172).

conditioned reflexes.* For, if it is assumed that the centre of the conditioned inhibitor forms inhibitory connexions with the centre of the *given* unconditioned stimulus, there is no reason for it to act inhibitorily on the centres of *other* unconditioned stimuli unless the two centres partially overlap. Pavlov bases his statement on Babkin's experiments concerning alimentary and acid conditioned reflexes, both measured by the same indicator—the secretion of saliva. But a great deal of evidence indicates that the relationship between these two groups of reflexes is complex, and far from being purely antagonistic. And so, if the conditioned inhibitor of the alimentary reflex does in fact inhibit an acid reflex or vice versa, this in our view only indicates that the alimentary and acid centres partially overlap, which is, for that matter, suggested by the identical nature of the two reactions. A genuine proof of Pavlov's statement would be the demonstration that, for instance, a conditioned inhibitor of an alimentary reflex inhibits the defensive conditioned reflex reinforced by stimulating the paw with an electric shock. But no one so far has demonstrated this.

From the foregoing it follows that conditioned inhibition may be very useful for a better understanding of the structure of various groups of reflexes and their mutual relations. Indeed, if we establish that the conditioned inhibitor, acting inhibitorily on the group of conditioned reflexes reinforced by the stimulus S_1, is capable of inhibiting the reflexes reinforced by the stimulus S_2, that is direct proof that both these groups of reflexes are partially allied, in other words that the unconditioned excitation caused by the stimulus S_1 and S_2 partially overlap. In this manner we can disclose the 'affinity' between various groups of reflexes. It would be extremely interesting to see whether the results of these investigations proved to be identical with those obtained from research into alliances and antagonisms among reflexes, pursued by the method we proposed in Chapter VII.

6

Surveying the mechanism of differentiation (in the widest sense of this term) we must distinguish between two mutually in-

* Vide Pavlov, *Conditioned Reflexes*. p. 77

dependent problems. One concerns the mechanism of plastic changes involved in differentiation, and we have attempted to solve this particular problem in the two immediately preceding sections of this chapter. The other, no less important, concerns the question of the configuration of centres of differentiated stimuli, and their physiological structure. At present *this* problem is still far from solution, and in consequence many phenomena in the field of differentiation must remain mysterious, and we can only explain them by hypotheses comparatively little supported by experimental facts.

In the present section we consider certain difficulties which we meet with in this field, and shall try to indicate a way of overcoming them.

To begin with, let us consider the differentiation of two related tones, two similar sounds, two different geometrical figures, etc. In all these cases we may imagine the cortical centres of these stimuli in a simple manner as homogeneous cellular fields partially overlapping, and though this scheme represents nothing but deliberate over-simplification, it can successfully serve for explanation of the mechanism of the above-cited examples of differentiation. But let us assume that we apply an external stimulus in such a way that it is alternately reinforced and non-reinforced by an unconditioned stimulus, or reinforced once in three, or once in four applications. After a time a differentiation will be formed, and it will be of such a nature that this stimulus produces either an active conditioned reflex, or an inhibitory reflex, in dependence on its order in successive trials. If we took the view that each time exactly one and the same cortical centre is excited, it would follow that this centre, being excited, sets up sometimes excitatory and sometimes inhibitory impulses, a deduction which is obviously quite untenable. However, such an assumption is not at all necessary. For in reality the external stimulus is not an isolated phenomenon, but acts on the cerebral cortex in space and time; concurrently with it a vast number of other extero-, intero- and proprioceptive stimuli are in operation, while the traces of stimuli which had acted earlier still remain in the cortex As we said in Chapter VI, all this complicated compound of stimuli

takes part in the elicitation of a conditioned reflex, and its various elements may acquire more or less significance in accordance with the conditions of the experiment. In the cerebral cortex this compound stimulus possesses a certain 'centre', i.e. a complex configuration of neurons, which it excites, and there is no doubt that this configuration is slightly different with each application of the stimulus, above all because each time other traces help to form the background of its action. So, in the concrete case under consideration, the excitatory configuration corresponding to the stimulus varies slightly, in dependence on whether the stimulus is applied after a reinforced or a non-reinforced trial, and this very fact may provide adequate basis for differentiation.

Approximately the same kind of situation arises when we are dealing with an inhibition of delay. As is known, this inhibition arises when the isolated period of the conditioned stimulus lasts several minutes instead of the usual duration of 15–30 seconds. In the initial phase of its action the conditioned stimulus evokes an inhibitory reflex, which in its properties is in no way different from other varieties of internal inhibition.

How should we conceive of the mechanism of this kind of inhibition?

If we assumed that during all the period of its isolated action the conditioned stimulus excites exactly the same cells in the cerebral cortex, we would find it quite incomprehensible that these cells could set up first both inhibitory and excitatory impulses, and then only excitatory impulses. However, we suppose that such an assumption would not correspond with the true state of affairs. For there is no doubt that a delayed reflex, like trace reflexes and reflexes 'to time',* arise because the animal discriminates between particular moments of the operation of the stimulus, i.e. here we are dealing with differentiation, but not of different similar stimuli, but different moments in the operation of one and the same stimulus. The moment immediately

* Trace reflexes are those which arise when the conditioned stimulus is reinforced not during its action, but some minutes after it has been withdrawn Conditioned reflexes 'to time' arise when the unconditioned stimulus is simply applied at identical intervals of time

preceding the unconditioned stimulus becomes an active conditioned stimulus, the more remote moments are differentiated and become inhibitory stimuli So we have reason to assume that the delay inhibition, like every other differentiation, occurs because of slight differences in the configuration of the centres of two similar stimuli, in this case the two periods of action of one and the same external factor. The closer the two moments of this factor are to each other, the finer is the differentiation, i.e. the corresponding 'centres' overlap to a greater extent. On the other hand, two remote moments of action of the inhibitory stimulus (e.g the beginning of its isolated action and the moment immediately preceding the presentation of food) are not very 'similar' to each other, and so they correspond to the relations which are observed in coarse and easy differentiation

So we consider that the best and, in our view, most probable model for inhibition of delay is a spatial one, in which the various moments of the action of the conditioned stimulus behave like all similar stimuli and, consequently, possess in the cerebral cortex configurations of neurons which are partially identical, but do not completely overlap. Speaking generally, we assume that the capacity which the cerebral cortex possesses of analysing purely temporal relations must be achieved with the aid of spatial mechanisms. But we have no idea of the kind of cortical structure we have to deal with, or of the nature and forms of its functioning. We can say with all certainty that the cerebral cortex does possess a sufficiently complex and rich functional equipment to analyse phenomena of the external world both in time and in space to exactly the same extent as does the psyche of the individual, but the nature of this equipment still remains a secret

However, it seems to us that there are categories of facts which provide certain indications of the line that should be followed in investigations into the above problem. These facts are known both in psychology and in physiology. In experiments with conditioned reflexes they assume the following form.

Rosenthal was the first to point out* that the differentiation of two frequencies of rhythmic tactile stimulation of the skin,

* Rosenthal, O S , *Trudy Lab Pavlova*, 1926, vol I, nos. 2/3, p. 141.

formed on one place, also applies to stimulation of other places without elaboration. Experimentation of this type was developed by Dolin,* who showed that the differentiation of two rhythmic stimuli of a given analyser (e.g. a metronome beating in different rhythms) not only *ipso facto* passes to other rhythmic stimuli of the same analyser (hissing, bells, etc), but even to the rhythmic stimuli of other analysers (e.g the rhythmic lighting of a lamp), i.e. the rhythm is, so to speak, abstracted by the animal from the other characteristics of the given stimuli.

Similar phenomena of transposition are found very frequently in the higher nervous activity of animals, and especially of man. Thus, knowing a certain tune, i e. differentiating it from other combinations of sounds, we recognize it as such irrespective of the key in which it is played; we recognize a given shape visually as such, independently of the part of the retina (and therefore of the visual area of the cerebral cortex) on which its image falls; if we have differentiated two definite shapes one from the other, their differentiation is preserved independently of their magnitude, etc. All these phenomena are regarded by certain psychologists as a main argument against the physiological approach to higher nervous activity, and as a proof of the 'bankruptcy' of physiology in this field None the less, we shall attempt to analyse them from the physiological aspect.

From the facts cited it follows that if differentiation has been elaborated to a particular property of a stimulus, or compound of stimuli, it can be transferred 'automatically' to other stimuli or their compounds furnished with this same property, even when these stimuli are acting on other analysers. So we must either assume that the conditioned connexion, formed or differentiated from others, passes on its copies, so to speak, to different areas of the cerebral cortex, which is hardly a very credible assumption and is difficult to justify physiologically, or else that the centres of stimuli are so constructed that they satisfy the possibilities of the phenomenon of transposition. Let us recall that, as a matter of fact, nervous impulses reaching the nerve cell do no more than provide impetus for its own activity, which by no

* Dolin, A. O , personal communication, also *Conference on Problems of Higher Nervous Activity*, 1937, p 17.

means consists in the faithful passing on of the messages which it has received. Bearing this in mind, we may assume that stimuli reaching a certain, even narrowly localized, spot in the given projection field of the cerebral cortex, are transferred to centres of 'higher orders', which react selectively only to definite characteristics of these stimuli. Thus the stimulus is, so to speak, broken up into its component characteristics and analysed in the literal sense of the word, to the degree and extent corresponding to the possibilities of the given cerebral cortex

Accepting the existence of such complicated 'many-storied' nerve centres in the cerebral cortex, we can easily explain the various cases of transposition with the aid of the mechanisms previously described. We take, for example, the above-described experiments by Dolin. The excitation produced in the cerebral cortex by beats of a metronome with a frequency of 120 to the minute is extremely widespread; all the characteristics of this stimulus that the animal is capable of seizing upon have their own separate cortical 'representation', and each of these 'representations' takes part in the general pattern of excitations caused by the action of the metronome beating at 120 to the minute The same metronome beating at 60 to the minute evokes an almost identical pattern of excitations, for only in the 'centre of rhythm' are other cortical elements excited. Hence, a conditioned reflex formed to the first of these stimuli is also generalized to the other. But if the metronome beating at 120 is constantly reinforced, while the metronome at 60 is not, then all other characteristics of the metronome except its rhythm lose their significance and are, so to speak, rendered indifferent (since the corresponding centres are bound up with the unconditioned centre both excitatorily and inhibitorily). On the other hand, those cell elements which correspond to the more frequent rhythm acquire exclusively excitatory connexions, while those which correspond to the less frequent rhythm acquire inhibitory connexions. If we now take a new rhythmic stimulus with a frequency of 120 beats to the minute and form a conditioned reflex to it (which is very easy to accomplish, since in the sphere of rhythm it overlaps with the corresponding fre-

quency of the metronome), this reflex is at once differentiated, since it somehow utilizes the same arrangements in the sphere of the rhythm centre as were formed in the differentiation of the metronome's frequency.

We must once more emphasize that the speculations presented in this section are of a provisional character, and serve only to help in an understanding of various forms of cortical analytic activity which cannot be conceived with the aid of simple and intuitive spatial models. We are, however, quite aware that they may be nothing more than working hypotheses, which may be overthrown by further experimental investigation.

Chapter X

Internal inhibition. 2. The basic properties of inhibition

1

The dynamic properties of internal inhibition (i e. the results of mutual interaction between concurrent inhibitory and excitatory conditioned reflexes as well as acute changes of inhibitory reflexes under the influence of various factors) have been investigated by the Pavlov school thoroughly and in great detail. This is due to the circumstance that Pavlov derived the basic laws of the functioning of the cerebral cortex very largely from experiments on internal inhibition, while the corresponding properties of the process of excitation were established by him rather *a posteriori*, on analogy with those mechanisms which had been established for inhibition For this reason, when we set about analysis of this field of phenomena we are able over a wide range to make use of an abundance of accumulated factual material, and to find in it a great deal of experimental evidence to support our argument

To ensure a greater clarity in our further exposition we shall once more briefly present the basic properties of internal inhibition, using the Pavlovian nomenclature only for their denomination, and not for their interpretation.

(1) If an inhibitory stimulus is followed by an active conditioned stimulus, the effect of the latter is temporarily reduced to a lesser or greater extent (so-called inhibitory after-effect). This diminution usually is the stronger and the more prolonged, the more times the inhibitory stimulus has been successively repeated (so-called summation or deepening of internal inhibition)

(2) Inhibitory after-effect does not usually appear in all its strength *during* the action of the inhibitory stimulus, nor *immediately* after its cessation, but at first increases for a period of from a few tens of seconds to 2–3 minutes, then gradually decreases over a period much more prolonged. (Experiments

by Kogan, Anrep, Ivanov-Smolensky, Kreps, Andreyev, and Podkopayev)

(3) Inhibitory after-effect depends on the quality of the excitatory conditioned stimulus applied after the inhibitory stimulus Given an identical 'age' and strength of excitatory conditioned stimuli, the degree of inhibition depends on their similarity to the inhibitory stimulus. The greater the similarity, the stronger and more protracted is the inhibition of the corresponding reflex. Conditioned reflexes to stimuli which are dissimilar to the inhibitory stimulus (e.g. which belong to other analysers) are inhibited to a much lesser extent. (Experiments by Krasnogorsky, Kogan, Anrep, Ivanov-Smolensky, Bieliakov, and Podkopayev)

The facts presented in paras (2) and (3) provide the main basis for the law of irradiation and the concentration of the inhibitory process.

(4) If the conditioned stimulus is applied during the action of the inhibitory stimulus or immediately after its cessation, it may give an effect either slightly reduced by comparison with the norm, or a normal, or an increased effect This last phenomenon is called positive induction

(5) The phenomenon of positive induction occurs most often with differential inhibitors. It depends on the stabilization of the differentiation and its subtlety (difficulty, tension) If the differential inhibitor is very firmly established or is not very similar to the conditioned stimulus, positive induction rarely occurs If the differentiation is fine and fresh, positive induction is obtained easily and is strong (Kalmykov). Positive induction occurs more readily during the action of the inhibitory stimulus than after its cessation (Podkopayev). It is manifested in a reflex both to a stimulus differentiated from the inhibitory stimulus, and to other conditioned stimuli.

(6) If immediately after the active conditioned stimulus (given without reinforcement) an inhibitory stimulus differentiated from it is applied, this stimulus yields a positive effect, which will be the stronger, the more similar the two stimuli are to each other (Pietrova, Podkopayev). This fact is regarded as a manifestation of the irradiation of excitation.

(7) Extraneous agents not particularly strong, acting concurrently with the inhibitory stimulus, or slightly preceding it, cause disinhibition of the inhibitory reflex, i.e they temporarily restore its positive response. The less established the inhibition and the greater the 'tension' it possesses, the more readily disinhibition occurs.

(8) An increase of excitability of the unconditioned centre or a general increase of excitability of the brain produces a disinhibition of not too firmly established inhibitory reflexes.

(9) Two inhibitory stimuli acting concurrently or in immediate succession sometimes have a deeper inhibitory effect than each of them applied separately, but sometimes, on the contrary, they mutually disinhibit each other and have a positive effect.

Our task consists in linking up, systematizing, and re-interpreting all this assemblage of facts.

2

As we said in the previous chapter, the non-reinforcement of the conditioned by the unconditioned stimulus leads to the formation of inhibitory connexions (in addition to the existing excitatory ones) between the centres of these stimuli, and, as the result, impulses delivered by the conditioned centre to the unconditioned centre act on the latter both excitatorily and inhibitorily. The greater the quantity of inhibitory connexions formed between the two centres, the stronger is the action of the inhibitory impulses opposed to that of the excitatory impulses, and the more reduced is the conditioned response. The action of the inhibitory impulses may be interpreted as a temporary rise in the threshold of excitability of the unconditioned centre, i.e. as a shift of its characteristic curve to the right.

We shall first consider the differential inhibitory reflex, as being the most carefully studied form of chronic inhibition, and in particular we shall analyse that stage of its formation when full differentiation has just been achieved, i.e. when the stimulus has begun to produce a zero effect Thus in this stage the conditioned reflex has become completely subliminal, not, however, because the excitatory impulses falling on the cells of the un-

conditioned centre have grown fewer (as is the case in a considerable weakening of the conditioned stimulus, vide p. 112), but because the threshold of excitability of these cells has been raised as the result of the inhibitory impulses falling on them. In this case the subliminal excitation of the unconditioned centre is very strong (i.e. close to discharge), since just so many inhibitory connexions, but no more, have been formed as are necessary to prevent a conditioned effect.

Let us consider what will happen if concurrently with such a reflex an excitatory conditioned reflex is put into action. The effect of such interaction is shown diagrammatically in fig. 11. Let us assume that the conditioned stimulus, when acting separately, i.e. against the background of a normal characteristic of the unconditioned centre, evokes a conditioned response of the height aa'. The inhibitory conditioned stimulus, before it was transformed into an inhibitor, evoked a reflex of the height bb', but in the present stage its effect is zero, since during its action the inhibiting impulses produce a shift in the curve of the characteristic of the unconditioned centre to the position $A_1B_1C_1$. If these two stimuli act concurrently the amount of excitation they supply to the unconditioned centre is

$$Oa+Ob=Oc,$$

while they will act against the background of the inhibitory characteristic $A_1B_1C_1$. As is evident from the diagram, their joint effect cc' will be far greater than the effect aa', of the conditioned stimulus applied separately, i.e. we shall have a typical phenomenon of facilitation.

Thus we are confronted with the classic phenomenon of so-called positive induction. As can be seen, it is the result of the summation of the conditioned excitatory reflex with the subliminal reflex, evoked by the inhibitory stimulus—a summation which in this case leads to facilitation. As the mechanism of positive induction is, as we see, in certain regards similar to the Sherringtonian 'immediate induction', and as the term 'induction' in the latter sense is rarely used of recent years, we propose to retain it to denote the above phenomenon, rejecting the adjective 'positive' as unnecessary.

Now we must consider whether the known properties of induction can be explained with the aid of the mechanism we have just described.

We have said before that the finer the differentiation, the stronger is the manifestation of the induction. This fact is easy to understand, since the more the centres of the two differ-

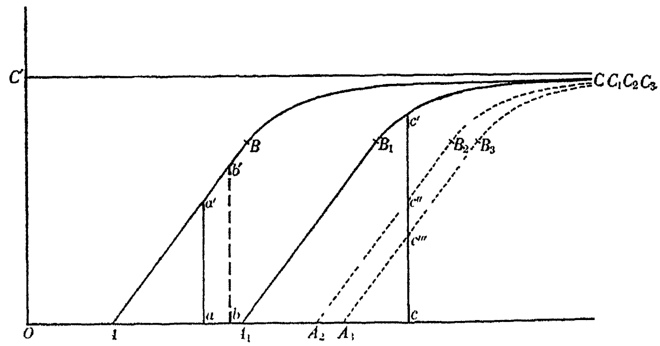


Fig 11. Interaction between inhibitory and excitatory conditioned reflex (induction). *Oa*, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by the active conditioned stimulus. *aa′*, effect of stimulus when applied separately. *ABC*, 'normal' characteristic curve of effector units of unconditioned centre *Ob*, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by the inhibitory conditioned stimulus. $A_1B_1C_1$, the characteristic curve of effector units of the unconditioned centre during the action of inhibitory reflex *Oc*, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by concurrent application of the active and inhibitory conditioned stimulus ($Oc = Oa + Ob$) cc', corresponding reflex response $cc' > aa'$ $A_2B_2C_2$ and $A_3B_3C_3$, characteristic curves of unconditioned centre during the action of more deepened inhibitory reflexes. cc'' and cc''' respective reflex responses when these inhibitory reflexes act concurrently with the given excitatory conditioned reflex $cc'' = aa'$. $cc''' < aa'$

entiated stimuli overlap, the more excitatory impulses are passed by the inhibitory stimulus to the unconditioned centre, and hence the better the conditions for summation and facilitation of both reflexes. On the other hand, with a coarser differentiation, when both stimuli have little in common, the inhibitory stimulus gives rise to few excitatory impulses, and so the facilitation is weak.

Similarly, the more elaborated and deepened the inhibitory reflex, i.e. the more the characteristic curve of the unconditioned centre is shifted to the right during the action of the inhibitory stimulus, the weaker of course is the facilitation, and the induction is less manifest Indeed, there is clear experimental evidence that an increase in the effect of the conditioned stimulus applied concurrently with the inhibitory stimulus is most readily obtained when the inhibitor is fresh, a fact that has long been known from Kalmykov's experiments.

It follows also from our analysis that, *ceteris paribus*, a strong stimulus applied as inhibitor produces a stronger induction than a weak one (since it passes more excitatory impulses to the unconditioned centre) and that weak conditioned reflexes acting concurrently with an inhibitory reflex produce a greater additional effect than do strong ones, since their effect is farther away from the top. While the first of these facts is well known, the second has not so far been checked.

We have also said that although induction is strongest during the action of the inhibitory stimulus, it can also be detected not long after its cessation. This fact is explained by the well-known evidence that the conditioned reflex (whether excitatory or inhibitory) outlasts considerably the application of the conditioned stimulus, always giving rise to a prolonged and strong after-discharge.

Finally, it has to be noted that, as Asratian established,* an increase in the reflex response during the action of the inhibitor, or immediately after its cessation, is to be observed in the case not only of the conditioned, but also the unconditioned reflex, this latter being increased by 10–15 %. This fact demonstratively indicates that although the inhibitory stimulus is not able to evoke supraliminal excitation in the unconditioned centre, nevertheless it causes a subliminal excitation in it, and this, summating with the direct action of the unconditioned stimulus, gives an increase in the latter's effect.

Now let us consider what happens if concurrently with or immediately after a fresh and not deepened inhibitory stimulus, not an active-conditioned, but another inhibitory stimulus is

* Asratian, E. A., *Trudy Lab. Pavlova*, 1941, vol. x, p 282.

applied. The situation that then arises is presented in fig. 12. The inhibitory stimulus s_1 sends an amount of excitation of the value Oa to the cells of the unconditioned centre, this excitation is subliminal because of the shift of the characteristic curve to the position $A_1B_1C_1$. The second inhibitory stimulus s_2 excites the cells of the unconditioned centre with the strength Ob, the characteristic of the cells being $A_2B_2C_2$.

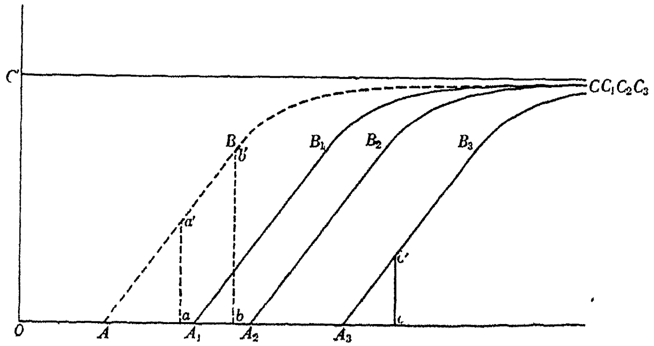


Fig 12 Interaction between two inhibitory conditioned reflexes (mutual disinhibition) Oa, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by the first inhibitory stimulus $A_1B_1C_1$, the characteristic curve of effector units of the unconditioned centre during the action of corresponding inhibitory reflex. Ob, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by the second inhibitory stimulus $A_2B_2C_2$, the characteristic curve of effector units of the unconditioned centre during the action of corresponding inhibitory reflex. Oc, the quantity of excitatory impulses delivered to the unconditioned centre in a unit of time by the concurrent application of both stimuli ($Oc = Oa + Ob$) $A_3B_3C_3$, the characteristic curve of effector units of the unconditioned centre during the joint action of both reflexes cc', corresponding reflex response As is seen, the concurrent application of two inhibitory stimuli results in this case in a supraliminal response.

If these two stimuli act concurrently, the amount of excitation set up by them will be equal to $Oa + Ob = Oc$, while the amount of inhibition jointly provided by them causes the curve of the characteristic of the unconditioned centre to shift still further to the right, to the position $A_3B_3C_3$ ($AA_3 = AA_1 + AA_2$). As can be seen from the diagram, with this position of the curve the excitation of the unconditioned centre becomes supraliminal

and is expressed by the magnitude cc'. So the concurrent application of two inhibitory stimuli may in sum have a positive effect, i e both the inhibitory reflexes may undergo mutual disinhibition.

In the example we have given neither of the two inhibitory reflexes was deepened to any great extent, the quantity of inhibition in each was only just sufficient to make them both subliminal. It is easy to see that a slight shift of either of these curves to the right causes the reflex response produced by the concurrent application of both stimuli to become subliminal, i.e. no disinhibition will follow. So, as can be seen, the mutual disinhibition of the two internal inhibitors is to be considered rather as a fairly rare fact, which can be obtained only with an appropriate relationship between the inhibitory and excitatory processes.

The experimental facts strikingly confirm the foregoing observations. The problem of the interaction between two internal inhibitors was investigated long ago,* and it must be said that while the majority of the authors stated that the processes of internal inhibition can only 'summate', in certain experiments the opposite result, i.e. mutual disinhibition, was also obtained. As these were rare cases, the view prevailed that disinhibition of the internal inhibitor by another internal inhibitor (in contradistinction to the action of external inhibitors) is impossible, and the exceptions to the rule were explained as disinhibition caused by fortuitously acting extraneous agents. But later, after the discovery of positive induction, the fact of mutual disinhibition of inhibitory reflexes was recognized, and experimental facts came to light which confirmed this viewpoint ('positive induction from inhibitor to inhibitor', as Siriatsky defined it).

Perhaps the most glaring case of mutual disinhibition of inhibitory reflexes is the shortening of the latent period of the

* The most important papers on the subject are: Nikiforovsky, P. M, *Trudy Obshch. Rusk. Vrachei*, Petersburg, 1909, vol LXXVI, Krasnogorsky, N. I., Diss , Petersburg, 1911; Patiokhin, S. I., Diss , Petersburg, 1912, Folbort, J. V , Diss., Petersburg, 1912; Leporsky, M I , Diss., Petersburg, 1911, Gorn, E. L , Diss., Petersburg, 1912, Ivanov-Smolensky, A G, *Trudy Lab. Pavlova*, 1932, vol. IV, p. 178, Siriatsky, V V , *Rusk. fis Zh.* 1926, vol. IX, no. 1.

excitatory conditioned reflex when acting in overlapping sequence with or in close succession to an inhibitory reflex. As is well known, conditioned reflexes dealt with by the Pavlov school are usually delayed reflexes, the first seconds of the action of the conditioned stimulus having inhibitory properties. This is because of the regular postponement of the reinforcement by 15–30 seconds after the conditioned stimulus is applied. Now the conditioned stimulus, given immediately after the inhibitor (or in overlapping sequence with it) as a rule has a shortened latent period, which can be explained (and which is so explained by various authors) as due to the disinhibiting action of one inhibitory reflex on another.

So, as we see, the Pavlovian positive induction consists in both the summation of the subliminal (inhibitory) reflex with the supraliminal conditioned reflex, causing an acceleration in the conditionally secreted saliva during the second period of action of the conditioned stimulus, and the summation of the two subliminal (inhibitory) reflexes, causing a shortening of the latent period of the excitatory conditioned reflex.

In strict connexion with the above-described phenomena is the fact of the disinhibition of the differential inhibitors, given immediately after the excitatory conditioned reflex (experiments by Podkopayev and Pietrova, p. 15). The conditioned stimulus causes an excitation of the unconditioned centre which greatly outlasts the period of its application. This excitation summates with the excitation of this centre caused by the inhibitory stimulus, and as the result, if the inhibitory reflex is not too deepened, the excitatory impulses obtain the predominance over the inhibitory ones, and disinhibition follows. The more similar the differential inhibitor is to the conditioned stimulus, i.e. the more excitatory impulses it passes to the unconditioned centre, the greater will be the joint effect of the two stimuli and the stronger, *eo ipso*, will be the disinhibition These relations are shown in fig. 13. So, as we see, the fact which from the viewpoint of Pavlov's theory was explained by reference to a mechanism exactly the converse of positive induction, namely, that of irradiation of excitation, is from our viewpoint based entirely on the mechanism of induction.

Indeed, in the summation of two concurrent allied reflexes it makes no serious difference in which sequence they act and to what extent they temporally overlap. In all such cases the

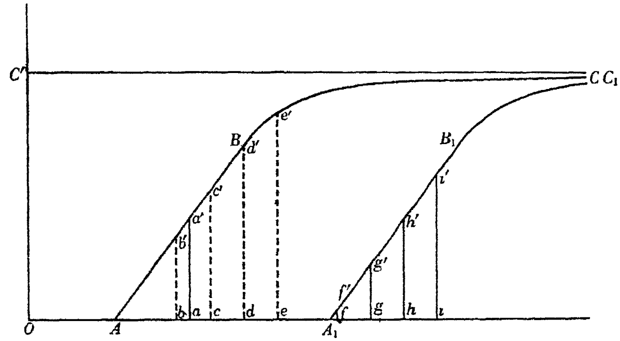


FIG. 13 Disinhibition of inhibitory conditioned stimuli applied immediately after the active conditioned stimulus (according to experiments of Pietrova and Podkopayev) *Oa*, quantity of excitatory impulses delivered to the unconditioned centre in a unit of time after the cessation of conditioned stimulus *ABC*, 'normal' characteristic curve of effector units of unconditioned centre *aa'*, the size of reflex response after the cessation of conditioned stimulus *Ob*, *Oc*, *Od*, *Oe*, quantities of excitatory impulses delivered to the unconditioned centre in a unit of time by inhibitory differentiated stimuli at various distances from excitatory stimulus. The more remote the given stimulus is, the less its centre overlaps with the centre of the conditioned stimulus, and the fewer excitatory impulses it sends to the unconditioned centre $A_1B_1C_1$, characteristic curve of effector units of the unconditioned centre during the action of inhibitory reflexes For the sake of simplicity it is assumed that all given reflexes yield the same amount of inhibitory impulses to the unconditioned centre, which is probably not true. *Of*, *Og*, *Oh*, *Oi*, respective quantities of excitatory impulses yielded to the unconditioned centre by inhibitory stimuli acting immediately after active conditioned stimulus ($Of = Oa + Ob$, $Og = Oa + Oc$, $Oh = Oa + Od$, $Oi = Oa + Oe$) *ff'*, *gg'*, *hh'*, *ii'*, respective conditioned responses evoked by these stimuli. As is seen, the more remote the inhibitory from the active stimulus the less the amount of its disinhibition The effect of the disinhibited inhibitory reflex can be either greater than the after-effect of the excitatory reflex ($ii' > aa'$) or equal to it ($hh' = aa'$) or smaller ($gg' < aa'$, $ff' < aa'$).

essence of the phenomenon of summation is exactly the same, and the insignificant quantitative differences are the result of differing intensities of the processes of excitation and inhibition at various moments in the course of both reflexes.

Just as our conception has enabled us to explain the mechanism of induction and the mutual disinhibition of internal inhibitors, so we can apply it for the interpretation of other properties of fresh and not too firmly established inhibitory reflexes. First of all, there is no difficulty whatever in explaining the fact of the disinhibition of an inhibitory reflex under the influence of the increased excitability of the unconditioned centre or of the whole brain Of course, these factors cause the characteristic curve of the unconditioned centre to shift to the left (cf fig. 6), so it is not surprising that they disinhibit those inhibitory reflexes which are immediately below threshold.

A similar mechanism of disinhibition operates in the case of concurrence of an inhibitory conditioned reflex with an extraneous reflex partially allied with it. Thus, in Bieliakov's experiments already referred to (cf. p. 116), an unusual sound of a trumpet evoked a strong aggressive reaction in the dog, and this led to a complete disinhibition of the inhibitory alimentary reflex. This is explained by the fact that the aggressive reflex causes *inter alia* a subliminal excitation of the alimentary centre, which summates with the similar excitation caused by the alimentary inhibitory reflex, resulting in a supraliminal response.

Here we arrive at the important problem of how we should explain all forms of disinhibition of inhibitory reflexes under the influence of extraneous agents. For it is clear that the case observed in Bieliakov's experiment is not typical, as usually disinhibition is obtained by the action of weak extraneous stimuli, whereas strong stimuli eliciting an orientation reflex of a defensive but not aggressive nature generally fail to cause disinhibition.

Unfortunately, although the phenomenon of disinhibition is among the best known of phenomena in the field of physiology of higher nervous activity, so far no research has been directed towards the elucidation of its mechanism The evidence that moderate extraneous stimuli have usually a double action, both inhibiting the excitatory reflexes and disinhibiting the inhibitory ones, leads to the assumption that one and the same stimulus may temporarily paralyse both inhibitory and excita-

tory connexions, and that the ultimate result of its action depends upon which of these connexions are affected more strongly Thus with the weak extraneous stimulus in action the inhibitory connexions between the conditioned and the unconditioned centre, being generally more fresh and unstable, are the first to be affected, and so the disinhibiting influence of such a stimulus prevails over its inhibiting action * But with stronger stimuli the inhibiting effect upon the excitatory connexions between both centres grows stronger and stronger, and therefore it more and more overshadows the disinhibiting effect Thus, with a very strong extra-stimulus the disinhibition of inhibitory reflexes is nil, not because it has no power of disinhibiting, but because it has a great power of inhibiting excitatory reflexes.

But, on the other hand, another mechanism of disinhibition must be taken into account, namely, that resulting from the generalization of excitatory conditioned reflexes. Let us assume that conditioned reflexes are formed in a dog to stimuli of various analysers, that one of these stimuli is transformed into an inhibitor, and that it is disinhibited by a weak extra stimulus It may happen very easily that this stimulus, being similar to one of the conditioned stimuli, evokes a subliminal conditioned response (the similarity being not so strong as to cause a supraliminal reflex). Now, if such a stimulus is applied concurrently with the inhibitory stimulus their subliminal effects will summate, and so the inhibitory reflex will be disinhibited. With stronger extra stimuli such facilitation is less probable, since they possess more foreign elements which will be in antagonism with the given conditioned reflex

The experimental evidence that very often excitatory conditioned reflexes are enhanced by the action of weak extraneous stimuli (vide p. 21) seems to testify in favour of the existence of the mechanism just described But, on the other hand, it must be realized that this fact may also be the result of dis-

* We do not believe, of course, that there is such a phenomenon as inhibition of inhibitory synapses, and therefore we have to assume that inhibition of an inhibitory reflex takes place in one of its intermediary excitatory relays

inhibition (in accordance with the first mechanism here presented) since, as we noticed above, the conditioned reflexes dealt with in Pavlov's school are more or less delayed, and an admixture of inhibition is always present in them. So, as we see, further experimentation is needed in order to disentangle all this problem.

The same phenomena which are manifested in the case of differential inhibition occur in experimental extinction and inhibition of delay at the corresponding stages of the process. The only exception is the action of the conditioned inhibitor (but *not* that of a compound stimulus consisting of the conditioned inhibitor plus the conditioned stimulus, which is nothing else than a differential inhibitor). In view of the fact that, as was emphasized in the preceding chapter, the conditioned inhibitor passes only inhibitory impulses to the unconditioned centre and passes no (or almost no) excitatory impulses whatever, it is obvious that it cannot either cause induction, or succumb to disinhibition. On the contrary, *its* activity is exclusively inhibitory; when it is added to conditioned stimuli it inhibits their effect, when it is added to the unconditioned stimulus it partly inhibits the unconditioned reflex. The difference between the properties of this inhibitor and other kinds of internal inhibition, which from the standpoint of the Pavlov theory cannot be satisfactorily explained, finds a complete explanation on the basis of the present conception.

3

Hitherto we have been considering that stage of formation of an inhibitory reflex in which exactly so many inhibitory connexions have been formed between the conditioned and the unconditioned centre as will balance the existing excitatory connexions, and no more. When the differentiation is very fine, i.e. when the number of inhibitory connexions cannot increase any further because of a considerable overlap of the centres of the two similar stimuli, this stage may last permanently. But in the event of a less fine differentiation, or an acute or chronic extinction, this stage is transient. So, in these cases the further repetition of the non-reinforced stimulus leads to an increase

of inhibitory connexions, which gain stronger and stronger predominance over the excitatory connexions.

What will be the effects of this deepening of the process of inhibition?

It is obvious that if to such a deepened inhibitory stimulus an active conditioned stimulus be added, instead of its effect being heightened it may be diminished, because, as the unconditioned centre is bombarded by the great number of inhibitory impulses, the curve of its characteristic will be shifted more to the right (see fig. 11). In other words, the inhibitory stimulus in this case will, so to speak, do more damage to the conditioned reflex with its inhibitory impulses than it will help it with excitatory impulses.

The general balance of activity of excitatory and inhibitory impulses falling on the cells of the unconditioned centre during the concurrent action of the inhibitory and excitatory conditioned stimuli is not a constant magnitude, but fluctuates in dependence on various, often even imperceptible, factors. As a consequence, in certain cases the result may be a reflex response greater than the normal response of the excitatory conditioned reflex, in other cases it will be smaller, and in still others it will be approximately equal That is what actually does happen (cf. the data of Podkopayev's experiments in the footnote to p. 18). It is well known that an increase in the effect of the conditioned stimulus applied in overlapping sequence with the inhibitor (i.e. positive induction in the Pavlovian sense of the term) by no means occurs always, and often it yields place to an insignificant diminution in the conditioned reaction. From the viewpoint of the Pavlov theory there is a fundamental difference between an increase and a diminution of the conditioned response, for in the first case we are dealing with positive induction, in the second with irradiation of inhibition. From our viewpoint the question is rather one of small fortuitous shifts in the relations between excitation and inhibition, and it would be a mistake to counterpose these two cases.

A deepening of the inhibitory reflex caused by an increase in the number of inhibitory connexions between the conditioned and unconditioned centre is, of course, manifested not only by

its influence upon excitatory conditioned reflexes. It is understandable that the disinhibition of such a reflex, whether as the result of external stimuli, or through an increase of the excitability of the centres involved, occurs with more difficulty. These facts are so well known that there is no need for us to dwell on them.

4

In the preceding sections we have analysed the influence of inhibitory reflexes on other active or inhibitory conditioned reflexes elicited concurrently or immediately after. Now we shall study phenomena which arise some time after the application of the inhibitory stimulus, i e. phenomena for which the Pavlovian terminology is 'the irradiation and concentration of inhibition', or (if we are to avoid imputing a definite mechanism to them) simply 'inhibitory after-effect'

We shall first consider those phenomena of inhibitory after-effect to which Pavlov gives the name 'irradiation of inhibition within the single analyser', i e which concern conditioned stimuli *similar* to the inhibitory stimulus (experiments by Krasnogorsky, Kogan, Ivanov-Smolensky, and others). In these cases the main mechanism involved in inhibitory after-effect is that of generalization. Indeed, as, according to our conception, generalization consists in a partial overlap of centres of similar stimuli, the extinction of the conditioned reflex to one of these stimuli must involve the partial extinction of reflexes to other stimuli, the extent to which this occurs being the greater, the closer the similarity of these stimuli to the stimulus originally extinguished. If extinction is chronic, then, of course, the secondary inhibition of other reflexes also has a chronic character. But if the extinction is acute, then the situation is rather different. For we know that such extinction undergoes regression, and so the secondary inhibition of other reflexes will also suffer regression

Let us consider this last case in more detail. We will assume that on the model of the experiments described in Chapter II (§ 2), we have elaborated conditioned reflexes to the stimulation of various places on a dog's skin, and that, after these reflexes have been stabilized, from time to time we carry out the acute extinction of one of them, while investigating the magnitude of

other reflexes at various intervals after the last application of the extinguished stimulus. Let us anticipate what will happen in such an experimental situation. First, if we test various conditioned stimuli at an *equal* interval after the extinction of one of them, we should find that their effects are the more inhibited, the closer the given stimulus is to the extinguished stimulus, i.e. the more their centres mutually overlap. Secondly, if the stimulation of some place on the skin is tested at *different* intervals after the extinction (but not at too brief intervals, in order to avoid the operation of induction), it should appear that the longer the interval the weaker will be the inhibition, because of the gradual atrophy of the fresh inhibitory connexions. It follows that the more remote the place on the skin is from the place originally extinguished, the more rapidly will the conditioned reaction to its stimulation return completely to normal; and the return to the norm will be slowest in the case of the reflex elicited from the place originally extinguished Thirdly, the more strongly one of the conditioned stimuli is extinguished (i.e. the more times it is repeated without reinforcement, and the shorter the intervals between the trials) the stronger should be the secondary inhibition of the other reflexes, and the slower should be their recovery. Fourthly and finally, inhibitory after-effect cannot be revealed in all its strength during the action of the inhibitory stimulus, or immediately after its cessation, because of the actual subliminal excitation of the unconditioned centre. It follows that in the first seconds and minutes after the cessation of the inhibitory stimulus the inhibitory after-effect increases with the gradual lulling of the excitation of the unconditioned centre.

Thus, as we see, all the facts upon which the Pavlovian conception of irradiation and concentration of inhibition is built up can be explained without its aid. They derive entirely from the principle of the formation of inhibitory connexions between the centres of the conditioned and the unconditioned stimulus when the first is not reinforced by the second, from the retrogression of those connexions when the inhibitory stimulus ceases to be applied, and from the partial overlapping of the centres of similar stimuli.

What is the inhibitory after-effect following the differential inhibitor? As we said in the preceding chapter, the centre of such an inhibitor is composed of two subcentres: one partially overlapping with the centre of the active conditioned stimulus, and the other not overlapping with it Between this latter subcentre and the unconditioned centre exclusively inhibitory connexions are formed, whereas the first subcentre is linked with it mainly by excitatory connexions But if the differential inhibitor is repeated several times in succession, a situation is created that in no way differs from extinction. The common part of the centres of the two differentiated stimuli produces fresh inhibitory connexions with the unconditioned centre, with the result that the active conditioned stimulus applied thereafter is partially inhibited (cf. Diegtiarova's experiments, described on p. 156). This inhibition will, of course, be transient, like all inhibition after acute extinction, and will be the stronger and the more permanent, the more the two centres overlap, i.e. the more fine the differentiation. Krasnorgorsky's and Bieliakov's experiments* completely confirm our deductions The authors showed that the finer the differentiation, the stronger and more lasting is the inhibition of the differentiated active conditioned stimulus applied after the inhibitory stimulus.

5

As we have seen, the principle of generalization of the inhibitory reflex completely explains the classic experiments of the Pavlov school concerning the secondary inhibition of stimuli similar to the inhibitory stimulus, i.e. 'irradiation and concentration of inhibition within a single analyser'. But we know that in the case of alimentary reflexes a strong inhibitory reflex (such as is obtained, for instance, by deep extinction of the conditioned reflex) often causes a diminution of the effect of conditioned stimuli from *other* analysers, and may even cause a partial inhibition of the unconditioned reflex Thus, after a deep extinction of the conditioned reflex it is sometimes observed that the

* Bieliakov, V V., Diss , Petersburg, 1911 (Pavlov, *Lectures*, vol. I, p 174) Krasnogorsky, N I , Diss , Petersburg, 1911 (Pavlov, *Conditioned Reflexes*, p. 154).

presentation of food fails to evoke salivation during the first few seconds of the act of eating Pavlov explains this as due to 'the irradiation of inhibition over the entire cortex' The question arises, how are these facts to be explained from the viewpoint of our conception?

It is well known that the alimentary centre (and possibly other unconditioned centres, too, but as to this point we have no clear evidence) is characterized by a certain *functional inertia*, which is manifested by the fact that when this centre is excited the salivary reaction considerably outlasts the actual duration of the stimulus, continuing sometimes for several minutes after the cessation of the act of eating Moreover, this excitatory after-effect even lasts much longer in a latent form, considerably facilitating the succeeding conditioned reflexes.* In the light of Lorente de Nó's conception of 'closed self-re-exciting chains of neurons' this phenomenon is completely intelligible. It is also clear that such long-lasting supra- or subliminal excitation can be immediately checked by the application of a strong inhibitory stimulus (external or internal)

The above-adduced facts concerning inhibitory after-effect are evidence that a similar mechanism of inertia is proper to the *inhibitory* alimentary reflex, i.e. that a strong and prolonged bombardment of the alimentary centre with inhibitory impulses has the effect of inhibiting it for a time, or of causing it to lose the acquired excitatory tonus And in this connexion, just as excitatory after-effect can be momentarily checked by the application of an inhibitor, so, conversely, the inhibitory after-effect is immediately abolished by giving the dog food, which completely 'washes out' the state of inhibition.

If such a prolonged state of inhibition in the alimentary centre is capable even of influencing the *unconditioned* reflex at the first moment of its action, its influence on alimentary

* This, *inter alia*, explains the soundness of the proverb: 'L'appetit vient en mangeant'. A phenomenon analogous to the meaning of this proverb is observed in many dogs When, after an almost 24 hr fast, the animal is taken to the experiment, the first conditioned reflex is usually very weak, and so it is often not even taken into account Only after the dog has eaten the first portion of food do the conditioned reflexes gain their 'normal' magnitude.

conditioned reflexes will be much greater, and they, too, as the result of this state will be inhibited to a greater or lesser extent. But as the secondary inhibition of reflexes to stimuli similar to the inhibitory stimulus occurs mainly in consequence of generalization, so, in order to observe the pure effect of inhibition caused by the inertia of the inhibitory process in the alimentary centre, we must employ conditioned stimuli dissimilar to the inhibitory stimulus, the best of all being those of other analysers. Indeed, we know quite well that secondary inhibition of the conditioned reflexes from other analysers is obtained much more rarely than from the same analyser as that of the inhibitory stimulus, that it is much weaker, and lasts a much shorter time This is quite understandable, if we bear in mind that in this case the inhibition of the reflex is due solely to the acute mechanism of inertia of the inhibitory process in the alimentary centre, whereas in the secondary inhibition of stimuli similar to the inhibitor, not only this mechanism, but the much stronger and more long-lasting mechanism of generalization comes into action.

So a diminution in the effect of alimentary conditioned reflexes to stimuli given a short time after a series of inhibitory stimuli may be due to two mechanisms, which will act in varying proportions according to the conditions of the experiment and the individual characteristics of the dog One is the manifestation of the plastic properties of the cerebral cortex, and depends on the formation of fresh inhibitory connexions between the conditioned and the unconditioned centre. The other is the manifestation of the excitability properties of the nervous system, and depends on the transient inhibition of the alimentary centre, being the result of the inertia of its activity, due most probably to its structure. We give below the similarities in the action of these two mechanisms, and the differences enabling them to be distinguished from each other.

(1) The mechanism of generalization of the inhibitory reflex and that of the inertia of inhibition in the alimentary centre act in one direction, and so their results summate.

(2) They both depend on the intensity of the inhibition, i.e. on the number of repetitions of the inhibitory stimulus.

(3) Whereas the mechanism of generalization embraces only stimuli similar to the inhibitory stimulus, and is featured by a definite gradient of inhibition according with this similarity, the mechanism of inertia causes, *ceteris paribus*, identical inhibition of all the conditioned reflexes, and also of the unconditioned reflex.

(4) The presentation of food wipes out the inhibitory after-effect caused by the mechanism of inertia, but it probably has hardly any influence on generalization of the inhibitory reflex

(5) A diminution of the reflex response as the result of the inertia of inhibition occurs only after the application of the inhibitory stimulus, whereas generalization of the inhibitory reflex is observed in both acute and chronic inhibition, i.e. is not necessarily bound up with the phenomenon of inhibitory after-effect.

(6) The mechanism of inertia to a large extent depends on the individual properties of the animal, and also on the group of reflexes being dealt with. It is very definitely to be observed in alimentary reflexes. The mechanism of generalization occurs in all animals and in all groups of reflexes.

If we take into account these two mechanisms of inhibition just described, we are able to understand a number of facts which hitherto have either been left unexplained, or have not received sufficient attention. We mention certain of them here.

It is clear that normally in acute extinction of the conditioned reflex two mechanisms jointly play a part: there is the formation of more and more numerous inhibitory connexions, and the increasingly strong inertia inhibition of the alimentary centre. For this reason the process occurs far more quickly than if only the mechanism of plasticity were in operation. On the other hand, chronic extinction, or differentiation (when the inhibited stimulus is applied among active stimuli, and never successively) depends exclusively on the mechanism of plasticity. Therefore a great incompatibility in the rate of the process in the acute and chronic forms of inhibition is often observed. We will illustrate this by the following example.

We compared the rate of acute extinction and differentiation in two dogs. In one dog (old) the differentiation was formed

slowly and with great difficulty, but the acute extinction of the conditioned reflex was each time obtained very readily and swiftly. In the other dog (young and lively) the same differentiation was formed much more rapidly, but the acute extinction was slower than in the first dog Taking into account the two mechanisms that may be involved here, it was easy enough to explain this disparity. The first dog, being very old, was much less capable of developing any plastic changes than the second Therefore elaboration of excitatory conditioned reflexes as well as the formation of chronic inhibitory reflexes occurred in the first dog slowly and sluggishly, while in the second dog all these processes ran a very rapid course But the first dog displayed a very great inertia of the alimentary centre in respect to both excitation and inhibition. So in this dog salivation after the act of eating was very protracted, and the inhibitory after-effect of each acute extinction was so strong that it influenced even the unconditioned reflex Therefore acute extinction occurred in this dog with great rapidity, while in the second it proceeded more slowly, as here only the mechanism of plasticity and not that of inertia was involved. By testing for inhibitory after-effect it was shown that in the first dog it affected reflexes to all the conditioned stimuli, whereas in the second it was manifest only in regard to stimuli similar to the inhibitor.

Many years ago the Pavlov school was greatly interested in the problem whether an acutely extinct conditioned reflex can be completely resurrected through the application of the reinforcing (alimentary) stimulus itself, or whether this stimulus, like the external stimulus, only temporarily disinhibits the extinguished reflex, but does not restore it. The discussion was vigorous, and opinions were divided. Finally, on the basis of Podkopayev's experiments, Pavlov came to the conclusion that the restoration of an extinct reflex through the unconditioned stimulus is only apparent, and that, if one waits long enough after the act of eating, the conditioned reflex proves to remain extinguished This even gave cause for Pavlov's enunciation of the following noteworthy opinion. 'The results of these recent experiments incline us more and more to believe that the inhibitory process arises in the nerve cells themselves

and not in the connecting path between those cells excited by the conditioned stimulus and those excited by the special unconditioned stimulus employed Otherwise it is difficult to reconcile the fact of the identical restorative action of the acid and the food.'*

None the less, it has to be pointed out that the results of various experimental investigations are in manifest contradiction to this 'decision' of Pavlov's. For instance, in Soloveychik's experiments, published much later,† the presentation of food quite unquestionably accelerated the restoration of the conditioned reflex, although this acceleration was not so considerable as when food was given together with the extinguished conditioned stimulus.

On the basis of the foregoing considerations one has no difficulty in disentangling this whole question and elucidating the sources of the discrepancies which are observed between various experiments. In those cases in which, because of the animal's individual properties, the inertia of inhibition played a large part in the extinction of the conditioned reflex, the presentation of food removed this state and *eo ipso* partly restored the conditioned reflex. On the other hand, in those cases in which the main cause of the extinction was the formation of inhibitory connexions, the actual presentation of food could not have any particular significance; only reinforcement of the extinguished conditioned stimulus by food could produce an effect. As the degree of the functional inertia of the alimentary centre can be determined in every particular case, the results of these and similar experiments could easily be foreseen.

6

Summarizing all that has been said in this chapter, we see that the effects of an inhibitory stimulus on the nervous system are numerous and many-sided.

First, the inhibitory stimulus evokes a *subliminal excitation* in the unconditioned centre, supplying it with both inhibitory and excitatory impulses. This excitation lasts only during the

* Pavlov, *Conditioned Reflexes*, p. 391

† Soloveychik, D. I., *Trudy Lab. Pavlova*, 1940, vol. IX, p. 270.

action of the inhibitory stimulus and shortly after it, and is the cause of such phenomena as induction and disinhibition. As the inhibitory reflex gets more and more fixed and the inhibitory impulses get predominance over the excitatory ones, both these phenomena are less and less clearly manifested, and the inhibitory reflex, instead of facilitating the active conditioned reflex, partly inhibits it. There is a purely inhibitory action in the case of the conditioned inhibitor.

Secondly, the application of an inhibitory stimulus (or, more strictly, the *non-reinforced* conditioned stimulus, no matter whether excitatory or inhibitory) initiates *plastic changes* in the cerebral cortex, consisting in this stimulus becoming more inhibitory (since its application causes the formation of new inhibitory connexions), while together with it other conditioned stimuli similar to it become more inhibitory. This property of the inhibitory stimulus explains certain of the phenomena of inhibitory after-effect, namely, those which come under the Pavlovian term of 'irradiation of inhibition over the given analyser'

Thirdly and finally, in certain groups of reflexes, e.g. in alimentary reflexes, the inhibitory stimulus may for a period cause a state of inhibition in the unconditioned centre, as an expression of the latter's functional inertia. This property is the cause of that part of the phenomena of inhibitory after-effect which comes under the Pavlovian term of 'irradiation of inhibition over the entire cerebral cortex'.

All these properties of internal inhibition combine in the most varied manner according to the conditions, with the result that in the majority of experiments the complete picture of the after-effect of the inhibitory stimulus is highly complex. However, knowing these properties and understanding their significance, we can often decipher the picture, and even anticipate it.

Let us try to do this for a number of different cases.

The most simple and typical kind of relations existing after inhibition is provided by the experiments of Ivanov-Smolensky (vide p. 13 and fig. 1). In these experiments (let us once more recall) extinction of one of the conditioned stimuli was conducted and the secondary inhibition of other, similar conditioned stimuli

was tested. As the diagram shows, inhibitory after-effect grew stronger during the first minutes after the cessation of the inhibitory stimulus, and began to dissipate only thereafter. The greater the similarity between the tested and the extinct conditioned stimulus, the deeper was the former's inhibition.

The fact that inhibitory after-effect did not manifest itself in all its strength immediately after the withdrawal of the extinguished stimulus but only after the lapse of several minutes, indicates that down to this moment induction was operating, i.e. interaction was present between the inhibitory conditioned reflex (outlasting the application of the corresponding stimulus) and the excitatory conditioned reflex evoked by the stimulus tested As is seen from the diagram, in this experiment facilitation was never very strong, and the response to a conditioned stimulus tested in no case exceeded its normal size. This fact is easy to understand. The conditioned stimulus subjected to extinction was always repeated without reinforcement several times in succession, till zero reaction was obtained. Of course, this procedure created quite favourable conditions for generalization of the inhibitory reflex. That it was really so is clearly seen from the fact that the more similar the tested stimulus was to the primarily inhibited stimulus, the stronger was its inhibition. Now, if the inhibitory conditioned reflex to the extinguished stimulus was generalized, it follows that the conditioned stimuli tested were not 'pure' excitatory conditioned stimuli, but had a strong admixture of inhibitory properties, these being the stronger the closer their resemblance to the primarily inhibited stimulus. Hence it is obvious that the concurrence of two such stimuli could not bring about a great facilitation, as it was not really the interaction of an inhibitory and an excitatory conditioned reflex, but the interaction of two inhibitory reflexes, one stronger and the other weaker.

It is also quite clear that the more remote the tested stimulus from the primarily inhibited stimulus, the less inhibitory it is, and the greater the facilitation it provides when applied concurrently with the extinguished stimulus. It is quite possible that if the author tested a conditioned stimulus completely dissimilar to the inhibitory stimulus (e.g. one acting on another

analyser) the true Pavlovian induction would have been obtained, i.e. the reflex response would be higher than normal The absence of data concerning the stimuli of other analysers makes it impossible also to determine the role of inertia of the inhibition in the above series of experiments If we could do this it would enable us to understand in full detail the whole of the experimental situation under discussion

To summarize: the effect of the conditioned stimulus applied immediately after the extinct stimulus (which gives 30–100 % of the norm) is the result of interaction between two homogeneous, partially inhibitory conditioned reflexes, facilitation in this situation is achieved the more readily, the less inhibitory the stimulus tested. The inhibition of conditioned reflexes elicited several minutes after the suspension of the inhibitory stimulus (amounting to 70–100 %) is chiefly the product of generalization of the inhibitory reflex, and therefore is the stronger, the greater the similarity between the tested and the inhibitory stimulus. The gradual dissipation of this inhibition depends on the retrogression of the inhibitory reflex caused by the fading of fresh and not fixed inhibitory connexions The role played in the described experiments by the inertia of the inhibitory process is unknown, because of the lack of appropriate experimental data

The experiments of Kogan* and Podkopayev† ran approximately the same course, and had results very similar to those of Ivanov-Smolensky We shall briefly analyse Podkopayev's experiments. Several conditioned reflexes to the stimulation of different places on the dog's skin were formed, then from time to time one of these reflexes was extinguished to zero (by applying the corresponding stimulus at 2 minute intervals without reinforcement), and the effect of the inhibitory stimulus upon the other conditioned stimuli was tested Curves based on the figures reported by the author are presented in fig. 14. As can be seen, they are largely analogous to those of fig. 1. It is worth

* Kogan, B A., Diss , Petersburg, 1914 (Pavlov, *Conditioned Reflexes*, pp. 157 et seq)

† Podkopayev, N A , *Collection of Articles in Honour of Pavlov's 75th Birthday*, Leningrad, 1924, p 297 (Pavlov, *Conditioned Reflexes*, pp. 209, 216)

drawing attention to the fact that the author applied the tested stimuli not only in successive but also in overlapping sequence with the inhibitory stimulus, and found that the conditioned response was in this situation even greater than when the stimuli were applied in succession This fact is quite clear, as facilitation during the action of the inhibitory stimulus is always

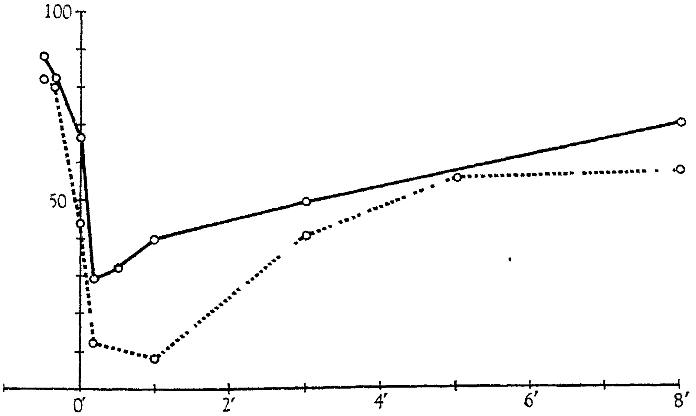


Fig 14 Inhibitory after-effect caused by extinction (on the basis of Podkopayev's experiments) Abscissa time elapsed after the last application of the extinct stimulus (in minutes) 0′ connotes the moment of cessation of inhibitory stimulus Ordinate· the size of conditioned response in percentage of normal response ---- inhibitory after-effect tested by stimulation of the place nearest to the one undergoing extinction (distance 1 cm) —— inhibitory after-effect tested by stimulation of the remote place (89 cm from the one undergoing extinction)

greater than after its cessation (see preceding section). It is also worth noting that in Kogan's experiments (also performed with tactile conditioned stimuli) the stimulation of the most remote place on the skin immediately after the stimulation of the inhibitory place sometimes elicited a more or less increased response. This effect and its happening precisely at the most remote place is quite intelligible, without needing any comment.

In Chapters II and III (pp. 18 and 46) we drew attention to the fact that, when acute extinction of some conditioned stimulus is carried out, another conditioned stimulus similar to it, applied

a couple of minutes after the extinguished stimulus (and so in a sense *instead* of this stimulus), gives a very strongly diminished reaction, whereas if it is applied *together* with the extinguished stimulus it gives an almost normal reaction. In the light of our present considerations this fact is quite clear. The conditioned stimulus similar to the stimulus primarily extinguished also acquires inhibitory properties, hence the reduction of its effect when it is given separately. But when this stimulus is applied concurrently with the stimulus primarily extinguished, there is interaction between the two reflexes resulting in a summation of the excitations they produce, this being manifested by a greater or lesser supraliminal response.

Quite different results from those of the previously described experiments were obtained by Kreps.* In his experiments, alimentary conditioned reflexes were formed in a dog to the stimulation of five different places of the skin along the body, while a differential inhibitor was formed of stimulation of a sixth place. Then, at various intervals of time after the inhibitor, one or another conditioned stimulus was applied, and its effect tested. The results are shown in the curves we have made on the basis of the author's statistical data (fig. 15). As can be seen from the diagram, these results differ from those of the previously cited experiments in the following respects. First, an effective and quite strong induction is to be observed Secondly, the inhibitory after-effect is weak, the conditioned reflexes are reduced to barely half. Thirdly, all the active stimuli (both close and remote in relation to the inhibitor) are inhibited more or less equally.

Are we able to explain the results of these experiments? We think we can. We draw attention to the fact that the differential inhibitor was applied only once, and so almost no conditions were present for a generalization of the inhibitory reflex. Thus the chief cause of inhibitory after-effect would be the inertia of inhibition in the alimentary centre, which accounts for the almost equal reduction of *all* the conditioned reflexes evoked after inhibitory stimulus, as well as for the comparatively small

* Kreps, E M , *Collection of Articles in Honour of Pavlov's 75th Birthday*, Leningrad, 1924, p 323 (Pavlov, *Conditioned Reflexes*, pp. 205–6).

amount of this reduction. As, according to our explanation, the conditioned reflexes elicited after the inhibitory reflex were 'pure' excitatory reflexes (generalization playing an insignificant part in these experiments), i.e. they supplied only excitatory impulses to the unconditioned centre, the existence of strong induction can be readily deduced If these experiments were

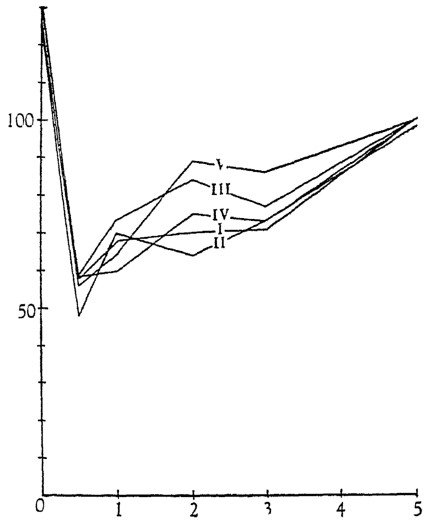


Fig 15 Inhibitory after-effect caused by differentiation (on the basis of Kreps's experiments) Abscissa: time (in minutes) elapsed after single application of inhibitory stimulus (tactile stimulation of a place on the shin) Ordinate the size of conditioned response in percentage of normal response. Curve I, stimulation of a place on the thigh Curve II, stimulation of another place on the thigh Curve III, stimulation of a place on the abdomen Curve IV, stimulation of a place on the chest Curve V, stimulation of a place on the shoulder

made to-day, it would not be difficult to test our explanation, for, if it is sound, then, first, the reflexes to the stimuli from other analysers would be inhibited to the same extent; secondly, the presentation of food to the dog should completely abolish the inhibitory after-effect; and thirdly, if instead of a differential inhibition the extinction of the conditioned reflex were used, then the results would be different and the gradient of inhibition in

relation to the distance from the extinguished stimulus would emerge more clearly.

Still other and yet more unexpected results were obtained in Andreyev's experiments.* This author formed alimentary reflexes to the stimulation of three spots on a dog's skin, along one hindlimb. A differential inhibitory reflex was formed to the stimulation of a fourth spot, situated farther up. As in Kreps's experiments, the inhibitory after-effect after a single application of the inhibitor was investigated. The author presented the results of these experiments in three graphs (fig. 16). As can be seen from the graphs, these results differ from the data obtained by Kreps, despite the fact that the course of the experiments was similar. First, the inhibition of all three points differed, but, conversely to the results of Kogan's, Podkopayev's and Ivanov-Smolensky's experiments, the point farthest from the inhibitory point was most strongly inhibited. Secondly, a quite strong induction occurred in the nearest point. Thirdly, the inhibition of the two nearest points was of an undulating nature.

Turning to analysis of these results, we assume that just as in Kreps's experiments, so in this case, generalization of the inhibitory reflex played no part. For it is not to be conceived that if it had been present the inverted relations between the inhibition of the near and the distant points could have occurred to such an extent. It follows that the curves which were obtained constitute the resultant action of induction and inhibitory after-effect, produced solely by the inertia of inhibition in the alimentary centre. As can be seen from the diagram, in the point farthest from the inhibitor induction is only feebly expressed; the action of the inhibitor amounts almost exclusively to inhibitory after-effect, which, be it noted (probably because of the individual properties of the dog), was far stronger than in Kreps's experiments. In the nearer point induction is more manifest, in the nearest point it is strong.

How is one to explain the different degrees of induction brought about by one and the same inhibitory stimulus? It seems that the cause of this phenomenon is to be found in the particular way in which these experiments were conducted.

* Quoted from Pavlov, *Conditioned Reflexes*, pp. 210–12.

First of all it has to be noted that the differentiation dealt with in these experiments was in general extremely fine, far finer than in Kreps's experiments. Moreover, immediately before the inhibitor the author applied the conditioned stimulus, which was tested afterwards in order to determine its 'normal' effect. So if a stimulus very close to the inhibitor was applied before-

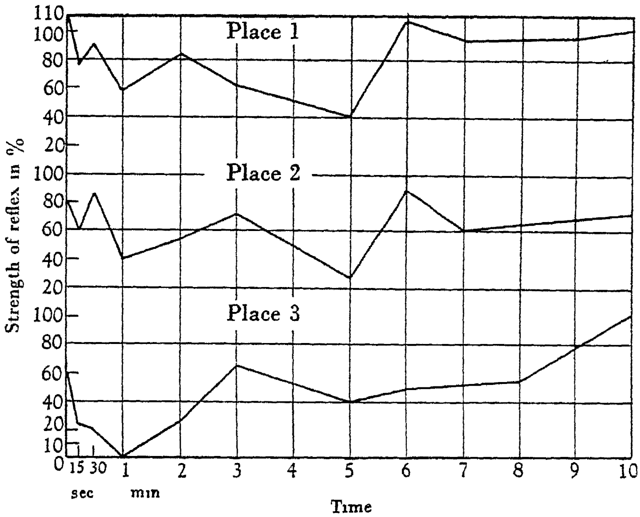


Fig. 16. Inhibitory after-effect caused by differentiation (on the basis of Andreev's experiments, taken from Pavlov, *Conditioned Reflexes*, p. 211, by permission of the Oxford University Press). Abscissa: time elapsed after the single application of inhibitory stimulus (tactile stimulation of the upper part of the thigh). Ordinate: the size of conditioned response in percentage of a normal response Graph 1, stimulation of a place on the leg nearest to the inhibitory place. Graph 2, stimulation of a place on the leg more remote from the inhibitory place. Graph 3, stimulation of a place on the leg still more remote from the inhibitory place.

hand its centre largely overlapped with the centre of the inhibitory stimulus, and therefore the excitation of this overlapping subcentre with reinforcement led to a strengthening of the excitatory connexions between it and the unconditioned centre. In consequence, if the differential inhibitor was applied in close succession it supplied a large number of excitatory

impulses to the alimentary centre, and so gave a strong induction. On the other hand, the application before the inhibitor of a remote stimulus, so that its centre was less overlapped with the inhibitor's centre, caused the inhibitory stimulus applied thereafter to send far fewer excitatory impulses to the unconditioned centre, with the result that in this case the facilitation was weaker. In other words, we assume that in each particular experiment we had to deal (owing to the special experimental conditions), not with an identical differential inhibitor, but with one sometimes more, sometimes less fine. But hence follows the simple deduction that this inhibitor gave sometimes less and sometimes more induction. One will realize that the acute struggle between the excitation and the inhibition could cause the undulations in the curves obtained.

It is rather more difficult to understand the fact that the inhibition at the most distant point was not only deeper than that in the two other points, but lasted much longer. The probable explanation of this fact is as follows: As we said, the stimulation of the inhibitory point after the nearest excitatory point causes a far greater amount of excitatory impulses to be sent to the unconditioned centre than follows from a stimulation of this point after the most distant point. As a result, in the latter case the unconditioned centre is more strongly inhibited than in the former, hence it can be inferred that the inhibitory after-effect will be stronger and will last longer.

It is obvious that in this series of experiments also all our suppositions could be very easily tested experimentally, and thus more profound analysis of the results could be achieved.

In conclusion, let us consider one other fact which from the standpoint of the Pavlov theory is rather unexpected, but which from the standpoint of the conception advanced in this book can easily be foreseen

Podkopayev* elicited an active conditioned reflex against the background of an inhibitory reflex, and obtained the following results· when the active conditioned stimulus was brought into action almost simultaneously with the inhibitor, its effect was more suppressed than when it began to act rather later.

* Podkopayev, N. A., *Trudy Lab Pavlova*, 1932, vol. IV, p. 354.

Ivanov-Smolensky obtained similar results.* Applying an active conditioned stimulus almost simultaneously with the differential inhibitor, he established that the latent period of the conditioned reflex was considerably prolonged (c. 15 seconds instead of 6 seconds); but if the active stimulus was brought into action only after the inhibitor had been acting for 15 seconds, the latent period, on the contrary, was shortened (to 3–4 seconds), and the effect of the reflex considerably increased.

Both these authors concluded from their experiments that immediately after the inhibitory stimulus is brought into action, the process of inhibition irradiates over the cortex, then swiftly concentrates, only to irradiate again after the cessation of the inhibitor's action, and then once more to concentrate, this time much more slowly. It is easy to see that the original irradiation and concentration cannot be justified, and is only one more label mechanically stuck on to an appropriate phenomenon.

Asratian† observed an analogous fact in his experiments. He investigated, *inter alia*, the influence exerted by the inhibitory stimulus on the magnitude of the *unconditioned* reflex, and established that if the unconditioned stimulus (the presentation of food) was applied after the inhibitor had begun to act, its effect was increased by 10–15 % (which, of course, was explained by positive induction), but if it was brought into action simultaneously with the inhibitory stimulus its effect was diminished. He did not attempt to explain this fact by reference to the original irradiation of inhibition as the previous authors had done, but simply stated that to him it was quite incomprehensible.

In the light of the present conception there is no difficulty in understanding these facts. As we have repeatedly emphasized, in the ordinary experimental conditions adopted by the Pavlov school, when the conditioned stimulus constantly precedes the presentation of food by 15–30 seconds, the beginning of its action becomes a strong inhibitory stimulus. This can be easily demonstrated if the conditioned stimulus is applied shortly after the dog has consumed its food, during the secretory after-effect. We

* Ivanov-Smolensky, A G , *Trudy Lab Pavlova*, 1932, vol. iv, p 208

† Asratian, E. A , *Trudy Lab. Pavlova*, 1941, vol. x, p 282.

then observe that this secretion stops at once (just as when the inhibitory stimulus is applied) and begins again only after a few seconds, as a conditioned secretion to the stimulus applied.

Now, if the beginning of the action of the active conditioned stimulus displays inhibitory properties, the beginning of the inhibitory stimulus differentiated from it must have a far stronger inhibitory effect, since the already formed inhibitory connexions are added to by new ones arising as the result of the non-reinforcement of this stimulus by the unconditioned stimulus. So we must distinguish two phases in the action of the ordinary differential inhibitor. The first, corresponding to the inactive (latent) phase of the excitatory conditioned reflex, is characterized by a strong fixation and a great predominance of inhibitory over excitatory connexions. In the second phase, corresponding to the active phase of the excitatory conditioned reflex, the fixation of the inhibitory reflex is much weaker, and there is a state of relative equilibrium between the inhibitory and excitatory connexions. So it is not surprising that if the excitatory conditioned or the unconditioned reflex is applied during the first phase of action of the inhibitor, the inhibiting influence of the latter will prove to be stronger than the facilitating influence, and as the result we get a diminution of the effect of the reflex and/or a prolongation of its latency. But if the positive stimulus is concurrent with the second phase of the inhibitory stimulus, then the balance is quite different, and, if the inhibitory reflex is not too firmly established, the facilitating predominates over the inhibiting influence.

It seems to us that the examples we have cited make it clear that with the aid of the present conception (which as a matter of fact involves nothing but the logical consequences of the general laws governing the central nervous activity) a large number of very complex and, seemingly, mysterious facts related to the effects of internal inhibition can be elucidated. The inadequacy of the Pavlov theory in this respect can be shown by his own words: 'Considerable difficulties arise, however, when a general rule governing the inter-relations between the two processes is looked for. Why is it that in some cases undulations are present, in others absent, why in some cases

is the spreading inhibition preceded by a positive induction, and why in others the latter is not apparent, etc.? Now we are still confronted with a series of unrelated facts deprived of all order.'* And yet we should not forget that the experiments we have discussed were conducted under the guidance of quite different ideas from those which now provide their explanation, and that from our viewpoint they lack a number of essential elements which would make their content more intelligible and would provide material for their more exact analysis.

ADDENDUM

On the Phenomenon of Sleep

In concluding this chapter, we must devote a few remarks to the problem of sleep, because of the fact that this phenomenon often accompanies internal inhibition, and is treated by the Pavlov theory as an irradiation of that process over the cortex and the subcortical ganglia.

We have already (Chapter III, §7) drawn attention to the fact that in experiments with conditioned reflexes situations arise in which the dog falls asleep, though there cannot be any hint of irradiation of inhibition (the application of weak conditioned stimuli, or food reinforcement of electrical stimulation of the skin). These facts overthrow the Pavlov theory of sleep in its own fortress, and so all the more compel us to seek a new interpretation of the phenomenon

Obviously, here it is not possible or even expedient to discuss the problem of sleep in all its extent and complexity. We shall only try to show that the sleep phenomena which emerge in conditioned reflex experiments can be considered from exactly the same point of view as those sleep phenomena which are encountered in other situations.

Taking into account the whole body of the facts related to sleep, we must note that they can be divided into two groups.

* Pavlov, *Conditioned Reflexes*, pp 214–15. As the English translation of this passage was not exact enough for our purpose, we have presented it in a revised form

The first group of facts points to the probability that sleep is essentially a subcortical phenomenon, which is controlled and regulated by definite centres situated at least partly in the hypothalamus. This is indicated by the evidence that the electrical stimulation of certain parts of the hypothalamus causes sleep, that in encephalitis lethargica we find pathologo-anatomical changes in this region, that sleep is connected with the excitation of the parasympathetic system (and inhibition of the sympathetic system), and that it has in general a close relation to the vegetative life of the organism. To all this we must add that the so-called emotions connected with the central excitation of the sympathetic system, such as anger, joy, alarm, are in antagonism to sleep, keeping the individual awake, and sometimes causing prolonged insomnia. All these data support the assumption of the existence of a 'sleep centre', probably largely overlapping with the subcortical parasympathetic centres. We must, of course, remember that we use the term 'centre' here in a broad physiological sense, not ascribing too narrow anatomical bounds to it.*

On the other hand, we have clear evidence, partly from our own experience, that monotonous surroundings and absence of external stimuli have a soporific effect This has been proved in extremely demonstrative fashion by Galkin and Speransky, who, having deprived dogs of three receptors (olfactory, visual and auditory), found that they slept unbrokenly for 23 hours a day and awoke only to eat and to evacuate. It is worth pointing out here that we have no evidence to show that the cause of the sleep thus evoked is to be found in the cerebral cortex. A dog completely deprived of the cortex, but not deprived of receptors, does indeed sleep longer than a normal dog, but incomparably less than the dogs in Galkin's and Speransky's experiments.† On the other hand, we know that by varying the external stimuli we can maintain a state of wakefulness, or can awaken a sleeping individual.

Of course, we do not know in the least what is the mechanism

* See in this context the interesting work of Nauta, W. J. H., *J. Neurophysiol* 1946, vol IX, p 285.

† Vide Asratian, E. *Physiol. J U.S.S.R* 1938, vol XXIV/1–2, p. 36

of the soporific effect of the absence of stimuli, and its relation to the activity of the 'sleep centre'. Nor can we explain how the action of variegated stimuli prevents or dispels sleep. But it is worth noting that while the emotions to which we have just referred take away the inclination for sleep and cause wakefulness, the action of external stimuli simply prevents sleep but does not eliminate sleepiness. This indicates that these two factors act on two different links of the process under discussion.

Taking the foregoing remarks into account, we must first and foremost note that in normal experiments with conditioned reflexes, conducted according to standard procedure, the animal is placed in conditions mostly favourable to its falling asleep. Quiet, the monotony of the environment, a certain restriction of movements, all largely conduce to sleep. It follows that the fact that many dogs fall asleep during experiments is not particularly surprising, and the important thing is to ascertain what conditions increase this tendency.

The fact that sleep comes more easily when only weak stimuli, or always one and the same stimulus, are applied in experiments, or when there are long intervals between the conditioned stimuli, or when the experimental chamber is acoustically isolated, is so obvious that it calls for no comment. Nor is it surprising that, on the contrary, variety in the conditions of the experiment, the application of variegated conditioned stimuli, the application of active conditioned and inhibitory stimuli alternately—all this dispels sleep and keeps the dog on the alert. But even more interesting are other less intuitive factors which came to light through the work of the Pavlov school, and which call for more detailed analysis.

Thus, it appears to be beyond all doubt that a peculiar kind of 'affinity' exists between the internal inhibition of alimentary conditioned reflexes and sleep, and that sleep often demonstratively arrives with delayed reflexes, when the action of the differential inhibitor is prolonged, etc. Obviously, we cannot explain this fact without special investigations which would clarify whether this phenomenon also occurs in less soporific situations than that of the typical conditioned reflex experiment,

whether it also occurs during the inhibition of defensive reflexes, etc. In any case, it must be noted that the deep internal inhibition of alimentary reflexes is an important 'event' in the emotional life of the animal, and therefore its connexion in one way or another with sleep should not cause surprise. It is a matter for future investigation to determine what this connexion consists in.

Just as unintelligible is the extremely interesting fact that the stimulation of a dog's skin with an electric shock, reinforced by food, frequently leads to the animal falling asleep. At present this is only experimental raw material, but it should provide a starting-point for further analysis.

Summarizing what we have said, we must state that the valuable experimental material collected by the Pavlov school in this field should undoubtedly be taken into consideration in any investigation into sleep. But the interpretation of this material must be along totally different lines from those hitherto followed. Instead of accepting the 'irradiation of inhibition', a phrase which tells us nothing and is not susceptible of further analysis, we should treat the phenomenon of sleep as a definite unconditioned process, and take the line of investigating what functions of the central nervous system are favourable or antagonistic to it. Only in this way shall we be able better to understand the sleep phenomena arising in conditioned reflex experiments and so contribute to our knowledge of the mechanism of sleep in general

CHAPTER XI

The dynamic stereotype and hypnotic phases

1

In point of fact, a great part of this book has been devoted to analysis of the *magnitude* of conditioned reflex response, and of its changes under the influence of various factors. Thus in Chapter VI we discussed the effect upon the magnitude of conditioned reflexes of the 'age' of a reflex, the strength of the conditioned stimulus, and the excitability of the nervous centres. In Chapter VII the changes of conditioned response under the influence of concurrent homogeneous and heterogeneous reflexes were examined. And finally, Chapter X was chiefly devoted to analysis of the changes of conditioned response due to the concurrent action of inhibitory reflexes

The present chapter will be in a sense supplementary to those mentioned, as it will deal with those factors influencing the size of conditioned reflexes which so far have not been discussed. Strictly speaking, this chapter will go beyond the scope of this book, since, as we shall see, the problems we now intend to raise cannot be analysed to the same extent as those discussed in previous chapters, because of either the complexity of these new problems, or the insufficiency of experimental material. However, we shall survey them mainly in order to point to their existence and in part to the possibility of their future elucidation.

2

Investigators engaged in the study of conditioned reflexes long ago drew attention to the fact that, if a dog has grown accustomed to certain conditions of experiments which are repeated day after day, even a slight change in these conditions causes sometimes considerable perturbations in conditioned reflex activity. We can treat such a change as a factor provoking an orientation reaction. Every experimenter working in the field of conditioned reflexes must bear in mind the existence of such

a reaction to every 'innovation', whether it be the application of a new stimulus, or a new arrangement of old stimuli; otherwise he runs the risk of a misleading interpretation of the facts obtained. Let us, for instance, assume that we are engaged in the study of the laws of summation of conditioned reflexes. To this end we apply concurrently two conditioned stimuli which hitherto have always been applied separately. This will undoubtedly cause a greater or smaller orientation reaction, which may distort the true effect of summation. Only when we eliminate this reaction shall we be able to analyse the results satisfactorily.

It was Soloveychik's experiments* that first indicated what great importance a constant order of applying conditioned stimuli has for the dog's conditioned reflex activity. In these experiments the conditioned stimuli were applied day after day for a long period at identical intervals and in precisely the same order of succession. It appeared that a change in the order of the stimuli in one experiment caused a strong diminution in all the conditioned reflexes for several successive days. Although this effect is not displayed in the same measure in all dogs, but is characteristic only for the so-called 'weak type of nervous system', it is none the less highly significant. Kupalov obtained similar results in his experiments.† This investigator, as we have already mentioned, created a 'mosaic' of positive and negative stimuli by the stimulation of various parts of the skin, and repeated these stimuli day after day in a definite order, applying positive and negative stimuli in turn. When the order of the stimuli was changed the conditioned reflex activity of the dog was disturbed The introduction of a new conditioned stimulus had the same effect.

A further step forward in this field was taken by Asratian‡ and Skipin § These investigators obtained the following result, which later was confirmed again and again. If a system of conditioned stimuli is repeated day after day at equal intervals

* Soloveychik, D Y , *Trudy Lab Pavlova*, 1928, vol. II/2, p. 61

† Kupalov, P S , *Trudy Lab. Pavlova*, 1929, vol III/2–3, p. 39; ibid, 1933, vol v, p 345; ibid 1933, vol. v, p. 383

‡ Asratian, E A., *Trudy Lab Pavlova*, 1938, vol VIII, p 1.

§ Skipin, G W., *Trudy Lab. Pavlova*, 1938, vol. VIII, p. 16

and in the same order of succession for a long period, and then, in one of the experimental sessions, instead of these stimuli one and the same stimulus is applied successively at the same intervals (a weak stimulus being the best for the purpose), the conditioned effect of this stimulus changes according to the stimulus it replaces. When it replaces a strong stimulus its effect is strong, when it replaces a weak stimulus the effect is weak, applied in place of an inhibitory stimulus its effect is almost zero. So in each case this stimulus produces a reaction not proper to itself but to the stimulus which it replaces.

From all this evidence we see that in conditioned reflex experiments each separate trial is not an independent event but forms a certain whole with other trials Pavlov called this fact the 'dynamic stereotype' of the cortical activity. It shows that not only are individual conditioned stimuli linked up with the corresponding unconditioned stimuli which accompany them, but that conditioned stimuli applied in the same order of succession are also linked up *among themselves*, and the course of the entire experiment possesses its traces in the dog's cerebral cortex, and unfolds like a well-known melody heard over and over again A change in the usual system of stimuli may lead either to a more or less strong orientation reaction, which violates the dynamic stereotype and completely upsets the normal course of reflex activity, or, on the other hand, it leads to the dynamic stereotype being maintained, and the animal reacts on the old lines, and contrary to the new order of things, as though it had not noticed any change in the experimental conditions.

Although of recent years the dynamic stereotype of conditioned reflexes has been investigated in great detail, it seems to us that its significance in the conditioned reflex activity is not so far adequately recognized, and many facts which are obviously its product are artificially interpreted with reference to other laws. By way of illustration we give the following examples.

In one of his works Ivanov-Smolensky* found that when two differential inhibitors were applied one after another at intervals of 5–7 minutes (i.e. at the customary intervals separating the

* Ivanov-Smolensky, G. P., *Trudy Lab Pavlova*, 1932, vol. IV, p. 148.

application of stimuli), the second inhibitory stimulus almost always proved to be disinhibited, while the positive conditioned stimulus applied at such an interval was usually more or less diminished (inhibitory after-effect). The phenomenon of disinhibition of the inhibitor coming second in succession appeared the more puzzling in view of the finding that it was not disinhibited when applied only 1 minute after the first inhibitor. The author could not think of any explanation for this fact, and only stated its existence.

We think the fact just described must be regarded as a pure product of dynamic stereotype. In hundreds of experiments the dog was made accustomed to a single application of the differential inhibitor between positive stimuli, and so it is not surprising that he reacted positively to the stimulus applied immediately after the inhibitor, even when it was another inhibitor.

Here is another example. Every worker who has studied inhibitory after-effect, and for this purpose has had from time to time to carry out an acute extinction of the conditioned reflex, knows how quickly the dog is trained to this process. After a time it is sufficient to apply the stimulus two or three times in succession at brief intervals and without reinforcement for complete extinction to follow. Here, too, we have a case of the animal growing accustomed to the procedure. It simply *differentiates* the situation in which a conditioned stimulus applied in succession at brief intervals is not accompanied by feeding, from the normal situation in which this stimulus is applied among other stimuli and at other intervals, and when it is regularly followed by the presentation of food.

The stereotype is also the explanation of the fact that a differential inhibitor, regularly applied in the same order and always between positive conditioned stimuli, evokes an increasingly weak and brief inhibitory after-effect ('it concentrates', to use the Pavlovian term) The dog gets accustomed to the procedure in which the inhibitor is always followed by a positive conditioned stimulus, so it is not surprising that corresponding changes are effected in the dynamic of its cortical processes.

Moreover, with the aid of the stereotype one can undoubtedly explain those 'inductive relations' between a 'pair' of stimuli differentiated one from the other, concerning which so much is written in various works on conditioned reflexes. If a positive and a negative conditioned stimulus are applied very often in turn, the result is the formation of a dynamic stereotype, so that the application of the inhibitor leads to an intensification of the effect of the positive conditioned stimulus and, on the other hand, the application of the latter stimulus with reinforcement improves the state of the inhibitor.

The Kupalov experiments cited above may serve as a classic example of this phenomenon. Kupalov applied tactile conditioned stimuli, positive and negative, in turn for a long period at identical 7-minute intervals. When thereafter a positive stimulus was followed at the normal interval by another positive stimulus, the effect of the latter was diminished; if, on the other hand, a negative stimulus was followed at the normal interval by another negative stimulus, the latter was disinhibited. If a negative stimulus was followed by a positive stimulus not after 7 but only after 14 minutes, it was observed that after 7 minutes there was a spontaneous secretion of saliva, while the conditioned reaction to the applied positive stimulus was diminished. If the interval between the negative stimulus and the positive stimulus was increased to 21 minutes, the effect of the latter was normal. Similarly, if two positive stimuli were applied in succession at a 14-minute interval, the effect of the second conditioned stimulus was also normal, and so on.

The investigator explained all the above facts by reference to inductive relations between the corresponding regions of the cerebral cortex (hence the term 'cortical mosaic'), and where there were longer intervals between the stimuli he accepted the existence of 'waves of positive or negative induction'.

It is easy to see that the relations he obtained are by no means 'natural' and permanent properties of the cerebral cortex, but were *imposed* on it. The positive stimulus applied 7 minutes after the other positive stimulus gives a diminished effect not as the result of any induction, but because of the

stereotype formed in the dog, it 'expected' a negative stimulus, i.e. it possessed an elaborated inhibitory reflex 'to time', which lowered the value of the positive conditioned reflex. For similar reasons the application of a negative stimulus 7 minutes after another negative stimulus causes its disinhibition, the application of a positive stimulus 14 minutes after a positive stimulus gives a normal effect, and so on

Summarizing, we must point out that the cerebral cortex gets 'attached' to it not only those experiences which we wish to instil into the animal, but also others which occur in the course of the experiment but are not taken into account by the investigator. So if, in experiments with conditioned reflexes, attention is not paid to the entirety of the experimental conditions, a number of 'laws' of the functioning of the cerebral cortex will be discovered which will be ascribed to its permanent functional properties, and not to the fact that the experimental dog has been *taught* to react in this and no other way. And so, if the possibility of the emergence of various, sometimes complex stereotypes, often such as the experimenter had by no means expected, is not borne in mind when carrying out work on conditioned reflexes, mistakes can be made and artefacts peculiar to this field may be obtained.

Many such artefacts are to be found in the existing factual material devoted to studies of conditioned reflexes. It would seem that the experimenters frequently act in more or less the same way as magicians, who, much to the surprise of the audience, take out of an apparently empty pocket an article which they themselves have hidden there. The only difference is that the experimenter seems to be just as surprised and amazed at his discovery as the audience, forgetting that he has found only what he had previously put into the experiment.

3

As we said at the end of Chapter II, there are definite types of perturbations in a dog's conditioned reflex activity which have been called 'hypnotic phases', since they arise *inter alia* in states of sleepiness, and also in neuroses Pavlov explained these perturbations by reference to the 'top capability of cortical

cells', and to 'top inhibition'. Let us try to throw some light on this phenomenon from our viewpoint

First of all, attention must be drawn to the fact that the phasic phenomena we refer to by no means arise so frequently as is assumed. In this respect there is some confusion, due to the following causes. As is well known, the Pavlov school distinguishes a phase of equalization at a low level, a phase of equalization at a high level, a paradoxical phase at a low or at a high level, an ultra-paradoxical phase, and a narcotic phase (when the reflexes are reduced, with the preservation of normal relations between them). It is obvious that the specified 'phases' exhaust all the possible relations between reflexes, i e. no matter what the magnitude of two reflexes, one following the other, it will always be possible to attach one of the foregoing labels to their mutual relations.

It is well known that if a dog is placed in a condition which for any reason departs from the normal, e g. if it is restless owing to the need to evacuate, is sick, is in any pain, is sleepy, etc., the conditioned reflexes become simply *irregular*, which is quite understandable, since the excitability of the centres involved may change from minute to minute But if we analyse such experiments in order to determine phasic phenomena we shall undoubtedly find these phenomena, since it will be possible to subordinate each section of the experiment to some phase or other. Thus we may obtain now the animal's normal state, and now a narcotic, paradoxical or equalization phase in various combinations, according to the fortuitous relations between any pair of successive reflexes.

So if we are to avoid being misled when analysing the experimental material we must attribute to particular phases only such changes in the magnitudes of reflexes as are of a more or less permanent character and are repeated in identical conditions consistently and regularly.

As for the causes of the emergence of the equalization and paradoxical phases, we have already discussed them in Chapter VII, § 3. We then pointed out that so-called supramaximal stimuli cause a defensive reaction, which collides with their conditioned response, and as a result this response may be lower than that

of the usually strong conditioned stimuli. If the excitability of the dog's defensive centres is increased for some reason, the defensive reaction may appear even to such stimuli as normally do not evoke it. Thus a bell of usual strength, which normally elicits a maximal conditioned reflex in the dog, gives a diminished effect because of the admixture of a defensive reaction. As the result the equalization or paradoxical phase is obtained. In laboratory practice dogs are met with which 'cannot bear strong stimuli' (Pavlov's expression), i e. they display a defensive reaction to them. Normal work with such dogs can only go on when the loud auditory stimuli are muffled.

Undoubtedly such a mechanism of phasic phenomena had its share in the production of neuroses in various dogs after the Leningrad flood in 1924, when the Institute of Experimental Medicine was flooded In certain animals this catastrophe raised the defensive excitability to such an extent that the strong conditioned stimuli became 'supramaximal' and elicited a considerably diminished conditioned response.* Possibly this mechanism also plays a part in other observed cases of phasic phenomena. For instance, various investigators have found that there is frequently some weakening of alimentary conditioned reflexes to strong stimuli when caffein is administered. It would be interesting to clarify whether this phenomenon occurs because of the admixture of a defensive reaction owing to a strengthening of the excitability of defensive centres, or for other reasons

The important question arises, are *all* the cases of phasic phenomena to be explained in this manner? In other words, are the phasic phenomena displayed in conflict neuroses, and sleepiness, due to an increase in the animal's defensive excitability, or have they another mechanism? Although it is probable that the first explanation is correct, we would also like to point out another possible interpretation of the inversion of conditioned reflexes in the above states, which must be verified in further investigation.

There is no doubt that in neuroses caused in the dog by the

* Vide, for example, Speranski, A. D , *Trudy Lab. Pavlova,* 1927, vol. II/1, p. 3

methods adopted in the Pavlov school, i.e by a 'collision of excitation and inhibition', 'overstraining of the process of inhibition', etc., the functional conditions of the alimentary centre are far from normal. If a hungry animal does not take the food presented, turns away from it, or displays ambivalence, its alimentary function is certainly greatly disturbed. That is not to be wondered at, if we consider that this collision of opposite processes affected this very function. So, without going into a more detailed analysis of the phenomenon, we must state that in experimental neurosis we have to do with a 'neurotic state of the alimentary centre', and must reckon with the circumstance that, at least in the experimental conditions, the activity of this centre is not normal.

From Wedensky's classic experiments it is known that an 'altered' part of a nerve conducts impulses not like a normal part, but gives an inversion of relationship between the strength of the stimulus and the strength of the reaction, i.e. we have to do with a phenomenon to some extent analogous with the phenomena above described. The facts discovered by Wedensky can be at least partially explained on the basis of the existence of a subnormal phase of nervous excitability, i.e. on the basis of the same mechanism as served Gasser for his conception of inhibition.

So we may assume that in certain rather pathological states of nerve centres they can react to a dense shower of excitatory impulses with 'inhibition', due to the subnormal phase of excitability caused by the first discharge of impulses. We have written the term 'inhibition' in quotation marks, since we consider that the mechanism of this phenomenon is so distinct from that of the inhibition we have been considering, that to avoid a confusion in terms it would be wise to give this phenomenon quite a different name (vide p. 71).

The mechanism we have spoken of would without any great difficulty explain the so-called phasic phenomena in conditioned reflexes A similar explanation was in fact given by Pavlov and Razienkov, when the facts were observed. The difference between our and Pavlov's view of these phenomena consists in the circumstance that whereas he localizes them in the centre

of the conditioned stimulus, in our view they take place at the synapses of the unconditioned centre.

We must also briefly discuss the narcotic phase, which Norkina has investigated in more detail.* On the basis of her own and previous experimental material she concluded that the narcotic phase is characterized not by a simple reduction in the conditioned reflexes, but by a far stronger reduction of reflexes to weak than to strong stimuli. In other words, here the relations are quite the converse of those which arise in the equalization or paradoxical phase. The narcotic phase is clearly to be observed in many states · in sleepiness caused by narcotics, in the satiation of the animal, etc. As we have said in Chapter VI, there is no difficulty in explaining this state. It is caused by a diminution in the excitability of the unconditioned centre, which diminution affects the conditioned reflexes to weak more than to strong stimuli (vide, fig. 6, p. 104).

* Norkina, L N., Diss , 1945 (unpublished)

CHAPTER XII

Conditioned reflexes of the second type

Dedicated to the memory of DR STEFAN MILLER, *killed by the Nazis This section of the physiology of higher nervous activity was elaborated in a common association with him of effort and thought.*

1

In the chapter on the plasticity and excitability of the cerebral cortex we said that the formation of a Pavlovian conditioned reflex, and also its internal inhibition (as well as inhibition of an orientation reaction) are not the only mechanisms of plasticity. The higher animals display other kinds of plastic changes, whose gradual recognition and investigation by objective methods is an important task of the physiology of higher nervous activity.

For over ten years the writer was associated with the late Dr Stefan Miller in the physiological analysis of plastic changes which do not come within the scheme of Pavlovian conditioned reflexes and must be regarded as a separate kind of plasticity. They are the same changes which the behaviourist psychology has studied for many years under the group term of 'habits'. In this chapter we shall present the chief results of our research in this field.*

The starting-point for our investigations was the fact, known from everyday life, from training experience and from the evidence of behaviourist psychology, that if an animal's motor reaction leads to a 'satisfying state of affairs' (Thorndike) then it tends to be repeated in the same situation, but if it leads to an 'annoying state of affairs', then it tends to be avoided We subjected these generally known and universal facts of animal

* Our main works on the subject include: Miller, S and Konorski, J., *C R Soc. Biol , Paris*, 1928, vol xcix, pp 1155, 1158, Konorski, J. and Miller, S , ibid 1930, vol. civ, pp 907, 911, ibid. 1934, vol cxv, p. 91; *Podstawy fizjologicznej teorii ruchów nabytych* (*Bases of the Physiological Theory of Acquired Movements*), 1933, Warsaw; *Przegląd Fiz. Ruchu*, 1933, vol. v; *Trudy Lab Pavlova*, 1936, vol vi, p 119, *J Gen. Psychol* 1937, vol xvi, p. 264; ibid. vol xvii, p 405, J. Konorski, *Przegląd Fiz. Ruchu*, 1939, vol. ix, p. 191.

behaviour to conditioned reflex investigation, analysed them in regard to their structure and, to some extent, in regard to their physiological mechanism.

The first variant of experiments of the conditioned reflex type used to illustrate these facts was as follows.

If an animal placed in a certain environment (e.g a dog at the stand) is from time to time provoked into the execution of some movement (e.g. raising the leg), and if this movement is reinforced by the presentation of food, after a time it appears that in this environment the animal 'spontaneously' performs the same movement without the application of the factors which originally evoked it. If this spontaneous movement continues to be reinforced by food, then it is fixed and is performed by the animal with the maximal rapidity and frequency (see below).

If the movement is reinforced in the given environment by the introduction of acid into the mouth, or by some other unconditioned stimulus evoking a defensive reaction, then the animal resists the provocation of this movement by performing an antagonistic movement. For instance, if the movement provoked is flexion of the leg, the animal resists every attempt at its provocation by extending the leg.

The manner in which a movement is provoked is quite unimportant. It can be caused either reflexively (e.g. by stimulation of the paw with an electric shock or by pricking until the animal is forced to bend its leg), or by mechanical means (e g. bending the dog's leg with the hand or by a special apparatus), or by waiting until the animal performs the movement in reaction to stimuli imperceptible to us (e g. the dog scratches, barks, etc).

From these experiments it is evident that unconditioned stimuli can be divided into two categories: those which by reinforcing the animal's movement cause it to perform the movement spontaneously, and those which, by reinforcing the movement, cause it to perform an antagonistic movement, i e. to resist the performance of the movement required. We call the first class of stimuli *positive* unconditioned stimuli, and the second class *negative* unconditioned stimuli.

Let us make a more detailed analysis of the phenomena we have to deal with in these experiments. If the flexion of the

limb becomes a signal for presentation of food or introduction of acid into the mouth, then, in accordance with the general rules of conditioning, the alimentary or acid conditioned reflex is formed to this movement, or, more strictly, to the proprioceptive stimuli engendered by it, this reflex will be manifested by the secretion of saliva and a motor alimentary or defensive reaction. But, as is evident from the experiments cited, we have also to deal with a new phenomenon which cannot be anticipated on the basis of those rules, in the case of reinforcement by food the animal comes to perform the movement spontaneously in a repetition of the situation in which it was originally reinforced, i e. in a sense it provides itself with a conditioned stimulus heralding the positive unconditioned agent, but if the movement is reinforced by the introduction of acid, on the contrary, the animal begins to perform an antagonistic movement, or, in more general terms, to prevent the operation of the stimulus involving the negative unconditioned agent.

We shall attempt to express these relations in schematic form.

We take s to denote the compound of stimuli representing the circumstances of the experiment, r is the movement of raising the limb, and the corresponding proprioceptive stimulus, $\sim r$ is the antagonistic movement, $\overset{+}{S}_u$ is the presentation of food, $\overset{-}{S}_u$ the introduction of acid, and R_u the secretion of saliva and the unconditioned reaction generally. $\rightarrow$ denotes unconditioned evocation of R_u, $\dashrightarrow$ denotes conditioned evocation of R_u or r, $+$ denotes 'accompanies'.

Expressing the course of the experiments above described in the foregoing symbols, we get:

$$
\begin{tikzcd}[row sep=large]
 & & & \begin{array}{c}\text{Conditioned reflex}\\ \text{first type}\end{array} & & \begin{array}{c}\text{Conditioned reflex}\\ \text{second type}\end{array} \\
\text{If} & s+r+\overset{+}{S}_u \arrow[d] & \text{then (1)} & \underbrace{s+r} \arrow[d, dashed] & \text{and (2)} & s \arrow[d, dashed] \\
 & R_u & & R_u & & r \\
\text{If} & s+r+\overset{-}{S}_u \arrow[d] & \text{then (1)} & \underbrace{s+r} \arrow[d, dashed] & \text{and (2)} & s \arrow[d, dashed] \\
 & R_u & & R_u & & \sim r
\end{tikzcd}
$$

Thus, if the compound of stimuli $s+r$ is reinforced by the stimulus $\overset{+}{S}_u$ eliciting the reaction R_u, then, first, the compound s plus r becomes a conditioned stimulus and begins to produce the reaction R_u, secondly, the stimulus s begins to evoke the movement r; if the compound of stimuli s plus r is reinforced by the stimulus $\overset{-}{S}_u$, eliciting the reaction R_u, then, first, the compound s plus r becomes a conditioned stimulus and begins to cause the reaction R_u, secondly, the stimulus s begins to evoke a movement antagonistic to the movement r.

It is easy to demonstrate that this new conditioned reflex $s\dashrightarrow r$, or $s\dashrightarrow \sim r$, has a completely different mechanism from that of the ordinary conditioned reflex, i e. that it is due to other plastic properties of the cerebral cortex. To do this one needs only draw attention to the following differences between them:

(1) The response of an ordinary conditioned reflex is always identical with the response of the unconditioned reflex reinforcing it, this being due to the very nature of its mechanism. As a rule the response of the new reflex is different, for it is a movement which was reinforced by the positive unconditioned stimulus, or a movement antagonistic to the one reinforced by the negative unconditioned stimulus.

(2) Ordinary conditioned reflexes are always of identical pattern, irrespective of the unconditioned reflex which serves for reinforcement, the new conditioned reflexes are of different patterns in dependence on whether the reinforcing stimulus is a positive or a negative unconditioned stimulus. In the first case its response is the movement provoked, in the second the corresponding antagonistic movement

(3) The response of ordinary conditioned reflexes may be any reaction of an organism, whether somatic or autonomic. As for the new reflexes, their effect is probably only a somatic reaction, and the pyramidal system seems to be an indispensable condition of their formation.*

* We suppose that it is this feature which gives the motor activity the stamp of 'voluntary activity' which it possesses Indeed, if an individual is capable of performing a certain function because it leads to the achievement of a positive unconditioned stimulus, or to the avoidance of a negative

Because of the foregoing differences we have called the ordinary conditioned reflexes with which the physiology of higher nervous activity has hitherto been concerned, *conditioned reflexes of the first type*, while the reflexes we are now discussing we have called *conditioned reflexes of the second type*. The conditioned stimulus which evokes a conditioned reflex of the second type will be called the *conditioned stimulus of the second type*.*

2

We shall analyse, in the first instance, the category of facts in which the animal's motor reaction is reinforced by a positive unconditioned stimulus (*the first variety of conditioned reflexes, second type*). The experimental procedure which we usually followed consisted in causing passive flexions of the dog's limb by means of a system of pulleys, reinforcing each movement with food. After a time, as soon as the dog is placed at the stand it begins to lift its leg without provocation. At first these active movements are awkward and slight, and occur rarely. They are accompanied by a general motor restlessness in the form of shifting from foot to foot and many other additional movements. But with suitable training (when the active movement of the limb is further slightly assisted and when movements only of a certain extent are reinforced) the raising of the limb grows more and more fluent, it occurs more and more rapidly, and with

stimulus, it is, viewed introspectively, 'voluntary'. If we were able in the same manner to change the blood pressure, thus getting rid of a head-ache, for instance, or to diminish the secretion of gastric juice, so eliminating heartburn or the pains of stomatic ulcer, these activities would be just as 'voluntary' as motor behaviour.

* In recent years various authors observed the difference existing between two types of conditioning in question and gave them appropriate names: e g Ivanov Smolensky (*Korsakov J Neuropath. Psychol.* 1928, vol xxxi/3, p. 229) distinguished 'conditiono-unconditioned reflexes' (our first type) and 'conditiono-conditioned reflexes' (our second type), Skinner (*J. Gen. Psychol* 1937, vol. xvi, p 272) distinguished type *S* and type *R* of conditioning, etc Hilgard and Marquis, in their excellent monograph *Conditioning and Learning* (New York, 1940), subjected both types to thorough analysis and called the first type 'classical conditioning' and the second type 'instrumental conditioning'. As it is difficult to foresee which terminology will be ultimately accepted, in this book I use those terms which we have introduced in 1928 (Miller and Konorski, *C.R. Soc. Biol* 1928, vol. xcix, p. 1155).

an increasingly brief latency. Finally, the dog begins to raise its leg immediately it is placed at the stand, and raises it again and again as soon as it has eaten the portion of food which reinforced the preceding movement. Thus the frequency of the motor response is regulated by the size of the portions presented. While eating, the dog never raises its leg. Also, the application of a firmly established and adequately strong alimentary conditioned stimulus of the first type causes inhibition of the active movements. A strong extraneous stimulus evoking an orientation reaction acts in the same way.

A parallel examination of the salivary and motor reaction shows that the dog learns the trick of lifting its leg almost exactly when this movement (as a proprioceptive stimulus) becomes an alimentary conditioned stimulus, i.e when it begins to cause a conditioned salivation. From this the important conclusion follows that there is a strict connexion between the elaboration of the active movements on the one hand, and the formation of the alimentary conditioned reflex of the first type to the compound of corresponding proprioceptive stimuli on the other.

If we cease to reinforce an active movement by food, the dog at first continues to perform this movement, doing so with the maximum frequency, while saliva is secreted copiously. Gradually the movements of the limb grow more infrequent, and the secretory reaction diminishes. Finally both reactions subside more or less coterminously (fig. 17). The same conditions which cause a revival of the conditioned reflex of the first type, namely a long period without raising the limb (which corresponds to the non-application of the conditioned stimulus), a pause in the experiment, the presentation of food, or the application of a weak external stimulus, also cause a revival of the animal's active movements. Their non-reinforcement leads to their more rapid extinction, in accordance with the general laws of plasticity. Thus, all the factors that weaken or abolish the conditioned reflex first type to a movement, regarded as a conditioned stimulus (external inhibition, extinction, the animal's strong satiation, etc.), also cause a transient or more lasting disappearance of the conditioned reflex second type. All the factors which

cause a conditioned reflex first type to a movement to reappear (regression of acute extinction, disinhibition), are also favourable to the manifestation of the movement itself. In other words, a movement acquired as the result of conditioning second type recurs when (and with certain exceptions only when) the compound of proprioceptive stimuli engendered by its performance plays the role of active conditioned stimulus.

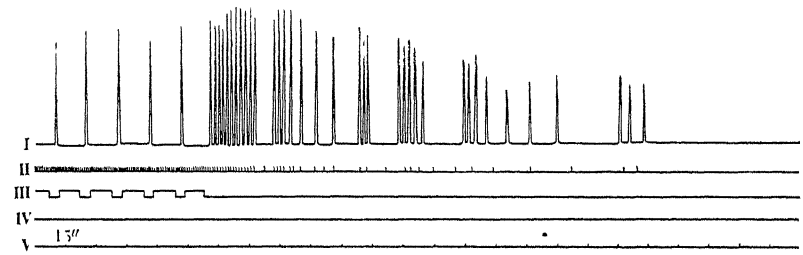


Fig. 17. Experimental extinction of the conditioned reflex second type (first variety) (semi-schematic) Conditioned stimulus of the second type, experimental environment Conditioned response of the second type, raising the leg. I, record of motor reaction of the second type. II, salivation (in drops). III, unconditioned reflex (act of eating) V, time (15 sec.) Explanations in text.

The rule we have just formulated also explains certain details of the formation of a conditioned reflex second type. When we raise the dog's leg to a certain height and reinforce this act by food, a conditioned reflex is formed not only to this concrete compound of proprioceptive stimuli, but, in accordance with the law of generalization, to all similar compounds. Hence it follows that the dog does not necessarily perform the exact movement which we tended to inculcate, but rather different movements (lower, partially with other limbs, etc), at the disposition of his motor system and within the 'field of generalization' of the given reflex. However, if we do not reinforce such movements, but only those of a definite height, definite shape, etc , gradually differentiation is formed, because only the *definite* compounds of proprioceptive stimuli are reinforced and in consequence only they become excitatorily conditioned. And it is this differentiation which effects the gradual shaping of an active movement in the form which the experimenter requires.

3

The disadvantage of the type of experiments just described is that the conditioned stimulus of the second type (in our case, the experimental environment: the dog at the stand, usually with a band round the leg, connected with an arrangement for recording movements, etc.), is a long-lasting stimulus acting throughout the experiment, and so it is rather indefinite and is difficult to control It presents the same situation as would arise if an ordinary conditioned reflex were formed simply to the experimental environment and not to particular single stimuli. And so our next task was to make the conditioned stimulus producing the active movements more definite and easier to operate —concretely, to make it a *sporadic* stimulus.

It is easy to understand that the key to the experiments just described is that for the animal in the experimental situation the performance of a certain movement is an *indispensable condition* of receiving food. In order to satisfy this requirement the experiments were conducted as follows: At normal, several-minute intervals we applied a definite indifferent stimulus (say the beat of a metronome), concurrently we mechanically flexed the dog's limb and presented food. (Such trials correspond to those moments in the action of the continual stimulus when we raised the dog's limb and presented food.) However, from time to time we applied the metronome separately, without raising the leg and without presenting food. (Such trials correspond to the moments in the action of the continual stimulus when the leg was not raised and the dog did not get food.) In such conditions the dog very quickly learns to raise its limb actively to each application of the stimulus, while the raising of the leg in the intervals (observed in the initial phase of the training) gradually disappears, not being reinforced.

But now let us see what happens if we try to form a motor conditioned reflex to a metronome, proceeding rather differently, namely, always combining the metronome with mechanical flexion of the leg, and reinforcing this compound with food. The result we get then is as follows. First, an alimentary conditioned reflex first type is formed to the metronome, the

dog displaying the secretory and the motor alimentary reaction (turning the head to the food table, licking the lips, etc.). Secondly, the dog begins to raise the leg actively with greater or lesser frequency during the intervals between the conditioned stimuli, these movements, lacking reinforcement, having a tendency to gradual extinction. But the active movements are not performed to the application of the metronome; in fact, rather the reverse is observed, as the movements are more or less definitely inhibited by this stimulus. There is not even a hint of any 'connexion' between the metronome and the raising of the leg.

The result we obtained is not difficult to understand. We reinforced the compound, consisting of the metronome and raising the leg, by food, and as the result each of these stimuli 'off its own bat' became an alimentary conditioned stimulus. Therefore the metronome began to cause a secretion of saliva, and the dog started to raise its leg actively, a visible sign that this movement, too, was becoming an alimentary conditioned stimulus. But the metronome, being a strong conditioned stimulus, not only failed to evoke the movement of raising the limb, but, on the contrary, inhibited it

Thus, if we apply only the compound, the metronome plus raising the leg, and reinforce it by food, the motor conditioned reflex *to the metronome* fails to be formed. To form this reflex one must return to the method previously described, i.e. must interweave the application of the compound, consisting of the metronome and the passive raising of the leg reinforced by food, with the application of the metronome separately without reinforcement, or, which amounts to the same thing, must prolong its operation, waiting until the dog finally does perform the required movement. In these conditions the dog soon begins to raise its leg actively to the metronome, the active movements during intervals gradually subside, and finally the trained movement recurs only to this stimulus, with a minimal latency.

From the foregoing data it follows that in order to form a conditioned reflex second type—metronome - - → raising the leg—it is not sufficient to have a simple concurrence of these two phenomena and to reinforce them by food. In order to form such a reflex, a situation has to be created in which the move-

ment is an *indispensable condition* of the dog's obtaining food, i e. it must be an indispensable complement of the given stimulus to the conditioned compound *

A more detailed explanation of this state of affairs is provided by the following experiments.

In a dog one or several alimentary conditioned reflexes of the first type are elaborated. Then to some new stimulus a conditioned reflex of the second type is formed, and, when this reflex is firmly established, both conditioned stimuli of the first type and of the second type are applied at the same experimental session. The following relations are then found: The conditioned stimulus of the first type as a rule does not provoke the second type motor response. But we need only cease to reinforce this stimulus with food, to get immediately the learnt motor reaction Similarly, if we apply a fresh and not too fixed differential inhibitor, then, too, the dog performs the trained movement to it, the inhibitor usually being disinhibited. Further, we may often observe that the second type motor response appears immediately after the dog has finished eating the given portion of food (fig. 18*a*).

These facts indicate that the movement acquired as the result of second type conditioning is by no means bound up with the stimulus to which it was formed, but may recur under the influence of other factors with which it had never previously been associated. On the basis of the above data it is easy to see that the performance of this movement is related with *the fall of active excitation in the alimentary centre.* This fall is caused either by cessation of the act of eating, or by the application of the alimentary conditioned stimulus without reinforcement, or of a fresh inhibitor. But it has to be added that the performance of the acquired movement (since this movement is an alimentary conditioned stimulus of the first type), causes a series of excitatory impulses to be sent to the alimentary centre, i.e. a fall of excitation in this centre is partially averted.

* We have discussed the whole of this problem in detail in the following works: Konorski, J and Miller, S , *Podstawy fizjologicznej teorii ruchów nabytych* (*Bases of the Physiological Theory of Acquired Movements*), Warsaw, 1933, and *Trudy Lab. Pavlova*, 1936, vol. vi, p. 119

Returning to the experiments we have been previously discussing, we see that the formation of a conditioned reflex second type to a metronome was obtained not because (or not only because) the passive raising of the leg was associated with this particular stimulus, but in the first instance because this stimulus was transformed into an alimentary conditioned stimulus of the first type, and then was inhibited (by not giving the dog food to it), i.e. it was turned into a signal of a fall of excitation in the alimentary centre.

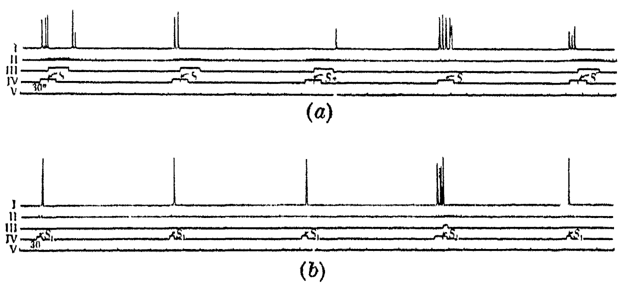


Fig. 18 Conditioned reflexes second type to sporadic stimuli (semi-schematic). I, record of the motor reaction of the second type (raising the leg) II, salivation (in drops) III, unconditioned stimulus (*a*, food, *b*, acid) IV conditioned stimuli V time (30 sec)

(*a*) Conditioned alimentary reflex second type (first variety). S, conditioned stimulus of the second type. $S+$, active conditioned stimulus of the first type $S-$, inhibitory fresh conditioned stimulus of the first type. To the stimulus S the dog raises leg, and obtains food To the stimulus $S+$ the dog exhibits 'pure' alimentary reaction without raising the leg. To the stimulus $S-$ the dog raises leg, and the inhibitory reflex is disinhibited. Note that raising of the leg sometimes occurs after eating.

(*b*) Conditioned defensive reflex second type (third variety). S_1, conditioned stimulus of the second type. S_2, active-conditioned stimulus of the first type. The dog raises its leg not only to the stimulus S_1 but to the stimulus S_2 and to the introduction of acid.

It is worth noting that exactly the same situation arises when a conditioned reflex of the second type is formed not to a sporadic stimulus but to the continual stimulus of environment. As is well known, giving the dog food at the stand leads to the formation of an alimentary conditioned reflex to the environment, i e. the dog salivates continually during the experiment and 'expects' food permanently. By applying some sporadic

stimulus reinforced by food (in this case a compound of proprioceptive stimuli engendered by raising the leg) the environmental stimulus is gradually inhibited, i e. the alimentary excitation which it evoked falls, whereas the sporadic stimulus acting against its background becomes an active conditioned stimulus. Thus the conditions for the formation of a conditioned reflex second type to environment are wholly fulfilled, according to the above-stated principles.

Summarizing, we come to the following conclusions·

(1) The motor response acquired by the mechanism of second type conditioning is by no means bound up with the stimulus to which it was formed, but may be elicited by other stimuli or factors.

(2) Active excitation in the alimentary centre caused either by a conditioned stimulus of the first type, or by the unconditioned stimulus itself (presentation of food), not only does not produce the acquired movement, but, on the contrary, has a tendency to inhibit it.

(3) A fall of excitation in the alimentary centre, such as occurs with non-reinforcement of the conditioned stimulus, cessation of the act of eating, or application of a fresh inhibitory stimulus, causes a recurrence of the acquired motor reaction.

4

Apart from the question of the intimate mechanism of the first variety of the conditioned reflex second type, the appearance and maintenance of this reflex depends entirely on the alimentary reinforcement A conditioned reflex of the second type, elaborated either to a continual stimulus, such as the experimental environment, or to a sporadic stimulus, subsides if it is not reinforced, just as does the conditioned reflex first type. This fact enables us to carry out exactly the same 'operations' upon conditioned reflexes second type (of the variety just described) as we can with conditioned reflexes first type.

Thus, if it is not reinforced by food, the reflex, metronome --→ raising of the leg, gradually disappears, just as does a conditioned reflex first type. If a stimulus similar to the conditioned stimulus of the second type be applied (e.g. a different frequency

of the metronome beat) it will by generalization evoke the same movement. But if the conditioned reflex second type to the previous frequency of metronome beat is constantly reinforced, whereas the new frequency is not reinforced, then differentiation follows, and the dog ceases to raise its leg to the new stimulus. Similarly we can form a conditioned inhibitor to a conditioned reflex second type, we can transform it into a delayed reflex, etc. In all these cases we have to deal with typical internal inhibition of a conditioned reflex second type, this being demonstrated by the possibility of disinhibiting the inhibitory reflex, by inhibitory after-effect, etc.

Experiments of this kind make it possible to examine simultaneously the course of the conditioned reaction first type—the secretion of saliva, and the conditioned reaction second type—the trained movement. The corresponding experimental evidence shows that these two reactions behave in approximately the same way. The relation between the two reactions largely depends on the individual properties of the animal We have had experience of dogs which exhibited a very exact parallelism in the two reactions; for instance, whenever the conditioned reflex second type was subjected to extinction both the acquired movement and the salivary reaction would disappear at exactly the same trial. But with other dogs different results were obtained. In certain cases the salivary reaction would undergo extinction (or differentiation) much earlier than the motor reaction, in other cases, on the contrary, the motor reaction became inhibited first. Taking into account the existence of subliminal conditioned reflexes, of which we have spoken in Chapter VII, we find these facts quite intelligible. In certain individuals, in which the 'salivatory centre' is of low excitability, while the motor centre is more excitable, at a definite stage of extinction the excitatory impulses sent to the first centre are not able to excite it supraliminally (owing to the rise in the threshold of its excitability, caused by the inhibitory impulses), whereas excitatory impulses sent to the motor centre are still able to evoke active excitation. In other dogs, in which the excitability relations are the converse, the motor reaction disappears first.

5

From the very structure of the conditioned reflexes second type it results that, with the aid of one and the same reinforcement (food), reflexes can be formed which will differ not only by their afferent part (as is the case in conditioned reflexes first type) but also by their efferent part For instance, to the beat of a metronome and the lighting of a lamp we can form reflexes consisting in raising the right foreleg; to the sound of a bell and a tactile stimulus—reflexes consisting in raising the hind left leg; to a tone and the smell of camphor—reflexes consisting in barking, etc. And so, while retaining the term 'group' for all conditioned reflexes (both first and second type), reinforced by one and the same unconditioned stimulus, we shall use the term *subgroup* for an assembly of conditioned reflexes of the second type, which differ from one another only by the stimuli, but possess the same response and the same reinforcement Reflexes belonging to a single subgroup we shall call homo-effectory, to different subgroups, hetero-effectory

If a dog has several elaborated hetero-effectory conditioned reflexes of the second type, then the individual motor reactions are by no means specifically bound up with the stimuli to which they have been formed, but may 'cross', i.e. they either replace other motor conditioned reactions or coexist with them. We have called phenomena of this kind 'cortical switching', and have established the conditions in which they appear.

They will be clear from the following experiments:

(1) An alimentary conditioned reflex second type consisting in raising the left hind leg to a tone was elaborated in a dog, while to the lighting of a lamp a reflex was formed consisting in raising the left foreleg. During the training of the first of these reflexes a band connected with an apparatus recording movements on a kymograph was fastened to the hindleg, and during the training of the second reflex it was fastened to the foreleg. After the elaboration of both reflexes it was established that when the band was on the foreleg, both the lamp and the tone evoked a movement of this leg; but when the band was on the hindleg both stimuli produced only the movement of the hindleg.

When there were bands on both legs, in the majority of cases the animal performed alternately both the acquired movements to each of the stimuli.

(2) In a dog running freely about the room an alimentary conditioned reflex of the second type (the beat of a metronome --→ barking) was formed. Then work on this reflex was suspended and in completely different conditions (at the stand behind a screen) we elaborated a reflex: tone --→ raising of the hindleg. When, in these conditions, after the establishment of this reflex we applied the metronome, the dog responded with an intensive raising of the hindleg, and only after a long period, when food was not presented, did it start to bark.

As can be seen from these experiments, the conditions in which the movement was elaborated and trained (e.g. the band on the leg, the given environment, etc.), predestine the appearance of this particular movement, irrespective of the stimulus which should evoke it. Similarly, a lengthy training of one of the formed movements with no training of the other, or a relatively greater ease of performing one movement, may have the effect that the movement will appear to a stimulus which 'normally' does not evoke it.

The experiments described in this section, as well as those in § 3, enable us to characterize the conditions governing the recurrence of movements acquired as a result of conditioning second type in the following manner. We can distinguish *determining* factors, which themselves do not evoke any movement, but designate what movement may be performed in a given situation, and *provoking* factors, which produce the movement designated by the determining factors. As can be seen from the experiments described, the determining factors are the external situation in which the particular movement was elaborated and trained, namely, the experimental environment, the band on the leg, etc Provoking factors are, as we have seen, those which cause a fall of excitation in the alimentary centre. To these belong *inter alia* the stimulus to which the conditioned reflex of the second type was elaborated, for this stimulus, as we have said, was at first an active conditioned stimulus of the first type, but then became partially inhibited. It must,

however, be noticed that this stimulus is not exclusively a provoking factor, but also to some extent designates the movement to be performed. This is best testified to by the fact that in the second experiment just described, the dog eventually 'recalled' that at the sound of the metronome it should bark, which it had never done at the sound of the tone.

It must be pointed out that we have it to a large extent in our own hands to strengthen or weaken the significance of these or other determining factors. For instance, if the experiments described in point (1) above are continued by retaining the bands round both foreleg and hindleg, and reinforcing by food only the 'correct' movements, we can finally make the sporadic stimulus a chief determining factor (even while it does not cease to be a provoking factor). On the other hand, if after some time the dog learns not to raise its leg when it has finished eating, or to some inhibitory stimulus, it is not because these factors have ceased to be provoking ones, but because in these circumstances the movement was not reinforced, and consequently underwent inhibition. But this does not mean that in these same conditions a new movement formed in the dog could not reappear.

6

So far we have been discussing the formation of a conditioned reflex of the second type, consisting in the animal learning to perform a movement which is an alimentary conditioned stimulus (of the first type) or is complementary to such a stimulus. The question arises, what will happen if the movement is, conversely, a hindrance to the receipt of food, i.e. is a conditioned inhibitor? In order to ascertain the answer we arranged an experiment on the following lines. A given stimulus (e.g. a metronome) being reinforced by food, the conditioned reflex first type is formed to it. Then from time to time we join to this stimulus a passive flexion of the leg, such combination not being reinforced. We shall soon find that the raising of the leg becomes a conditioned inhibitor, i.e. when applied concurrently with the metronome it inhibits the secretion of saliva to this stimulus. But in addition, more or less at the same time, the animal begins

to resist the passive flexion of the leg by actively extending it. After a time the animal performs an active movement of extension to the metronome itself, this movement increasing in strength with every attempt at mechanical flexion. Finally, the movement of extension becomes so strong that we are almost able to raise the animal into the air by its extended limb, and it can easily be recorded with the aid of a sprung floor.

Thus, contrary to the reflex previously described, in this case the *provoked* movement is not a conditioned stimulus, but a conditioned inhibitor, whereas the *formed* movement is not the one provoked, but a movement antagonistic to it. We shall classify such reflexes as the *second variety of conditioned reflexes second type.*

Despite the above-mentioned difference in the manner of forming the second variety of conditioned reflexes second type as compared with the first variety, when the reflex is already formed, its properties are entirely identical. For instance, its response may be replaced by another motor response, in accordance with the principle of switching, or may be brought about by non-reinforcement of the conditioned stimulus of the first type. We can also extinguish, differentiate, and delay such a reflex, just as we can the first variety of conditioned reflexes second type.

By inhibiting this variety we may disclose one very interesting and important property of motor conditioned reflexes which escapes our observation in the first variety. Let us assume that we have a well elaborated reflex· metronome ---→ extension of the limb, and that we inhibit it by extinction or differentiation. Then we find that at a certain stage of the process the dog's behaviour changes as follows: the limb which hitherto has appeared to be fastened firmly to the floor is gradually relaxed, the dog ceases to press its paw against the floor, and begins to perform a *flexion movement* This indicates that when the conditioned reflex of the second type is transformed from an excitatory to an inhibitory one, an antagonistic excitatory reflex simultaneously arises. This fact confirms the existence of reciprocal relations between the antagonistic motor cortical centres. We shall discuss its significance later.

7

We turn now to the analysis of *defensive* conditioned reflexes of the second type, i e. those formed with the aid of reinforcement by an unconditioned negative stimulus. At first we shall study, not the reflex we discussed in § 1, when the given movement signalizes the negative stimulus, but the reflex arising when the movement is a conditioned inhibitor. The advantage of such a sequence will be evident later.

Then the course of the experiment is as follows. The external stimulus (say the metronome) is reinforced by a negative unconditioned stimulus, e.g. the introduction of acid into the dog's mouth. The conditioned reaction consists in the secretion of saliva and making rejecting movements with the mouth. When this reflex is formed, from time to time we provoke a passive flexion of the limb in association with the metronome, and in these trials the acid is not introduced. After a comparatively short time a reflex is formed in which the dog itself raises the limb to the metronome—a movement saving it from the introduction of acid This reflex is soon established so firmly that for weeks and months the dog goes on raising its paw to each application of the metronome, even though the reflex is never reinforced (for the metronome is reinforced only when the dog *does not* perform this movement, which occurs very rarely). We call this reflex the *third variety* of conditioned reflexes second type (fig. 18*b*).

In this variety also, as in those previously described, the acquired movement is not specifically bound up with the stimulus to which it was formed. Thus, if we have previously formed an acid conditioned reflex first type to a number of stimuli, and then have formed a defensive conditioned reflex second type to one of them, the application of any of these stimuli evokes an identical reaction, even though previously this stimulus has never been associated with this movement. Similarly, if we take a new stimulus and reinforce it by the introduction of acid, as soon as the acid conditioned reflex of the first type is formed to it a conditioned reflex of the second type immediately appears in the form of raising the leg. The acquired

movement is also performed to the unconditioned negative stimulus itself, if this stimulus is not too strong.

So we see that whereas in the case of *alimentary* reflexes of the second type the factor producing the animal's movement (i.e. 'the provoking factor') is a fall of excitation in the unconditioned centre (caused by discontinuing the unconditioned stimulus, by extinguishing the conditioned stimulus, etc.), in *defensive* conditioned reflexes of the second type the factor is, on the contrary, a *rise* of excitation in the unconditioned centre, caused by the application of an active conditioned stimulus, or of an unconditioned stimulus. As we have said, the dog's performance of a movement which is an effect of an alimentary conditioned reflex second type averts a fall of excitation in the alimentary centre by providing it with new excitatory impulses. On the contrary, performance of the movement which is an effect of the defensive conditioned reflex second type averts a rise of excitation in the defensive centre. Indeed, as this movement is a conditioned inhibitor, it provides the unconditioned centre with pure inhibitory impulses.

Further experiments showed that if we reinforce the new stimulus by another unconditioned negative stimulus, e.g. by puffing air into the ear, the animal immediately begins to 'defend itself' against this stimulus also, performing the movement which saved it from the introduction of acid into the mouth. Therefore the defensive reflex second type is not bound up with the *definite* unconditioned defensive reaction with the aid of which it was formed, but is 'generalized' to heterogeneous reflexes, and so appears to conditioned stimuli of other defensive reactions.

We would like briefly to discuss the above property of reflexes belonging to this variety, a property never met with in other conditioned reflexes, and consisting in their being virtually inextinguishable, though ostensibly they are not reinforced in any way. The unusual character of this property must strike anybody who knows how rapidly the alimentary conditioned reflex second type subsides when it ceases to be reinforced, and who knows, too, that the simple association of passive flexion of the leg with the metronome (without the 'threat' of introduction of acid) does not lead to the formation of a motor reflex, though

this association be applied hundreds of times. In other words, work on conditioned reflexes of the second type brings out the fact that the dog does not perform any of his movements 'disinterestedly', so the fact that the particular reflex in question is maintained permanently, without any reinforcement, is all the more surprising.

There is ample evidence to show that the movement which is a conditioned defensive response of the second type continues to preserve a definite defensive character even after hundreds of trials without reinforcement. This is proved by the fact that if we introduce acid into the mouth or puff into the ear at the beginning of the experiment, for that experimental session, and also for some days after, the dog raises its leg to the conditioned stimulus more vigorously and with a briefer latency than usually. Moreover, it is sufficient to take a new stimulus and begin to reinforce it by a negative unconditioned stimulus, for the dog immediately to perform the acquired defensive movement to this stimulus. So the dog remembers that this movement saves it from the negative stimulus, and performs the movement for this very 'purpose'. But we have no evidence on which to assume that the defensive nature of the given movement remains specific, i.e that it is in some way bound up with the negative stimulus to which it was originally antagonistic. For, on the one hand we know that when the conditioned reflex second type: metronome ---> raising the limb (in which this movement defends the dog against the introduction of acid), is formed and fixed, we see no trace of a specific defensive reaction to the metronome in the form of a secretion of saliva, or rejection movements of the mouth On the other hand, as we have said, the dog performs the movement as defence against any negative stimulus Therefore the dog's defensive movement is not of a *specific* character, but the animal raises the leg in order to avert some undefined negative factor.

The conditioned reflex of the second type just described is best maintained when the applied conditioned stimulus is sufficiently strong (for these purposes an auditory stimulus is the best), and when this stimulus is interrupted as soon as the dog raises its leg. So one gets the impression that this very cessation of the conditioned stimulus is a peculiar form of 'reinforcement'

of the reflex, the more so as, if this stimulus continues to act over a long period, the dog 'in panic' raises its leg again and again in rapid succession for a very long time.

A sufficiently strong auditory stimulus possesses to a larger or smaller extent latent properties of the stimulus of defensive reaction, and in favourable circumstances (and, therefore, precisely in the case of its reinforcement by a negative unconditioned stimulus) these are enhanced (cf. p. 119). Therefore, whether because it is to a slight extent a negative stimulus and this negative quality has grown as the result of an alliance of all the defensive reflexes, or whether because this quality has been entirely imparted by conditioning, such stimulus *is* in any case a negative one, and therefore its cessation constitutes a certain type of unconditioned event which may serve as reinforcement for some conditioned reflex (just as withdrawal of food is also a reinforcement).

In appropriate experiments we were able to establish that if a place on the dog's body is stimulated not very strongly with an electric shock, and this stimulus ceases simultaneously with raising the dog's leg, the animal learns to raise its leg as soon as the current is applied. It is easy to see that there is only an insignificant difference between the two phenomena we have described. In the latter case the dog raises its leg in order to rid itself of the electric shock, in the former case it does so in order to rid itself of the stimulus which heralds the negative unconditioned stimulus.

But the following interesting circumstance is worthy of attention. If, after an acid conditioned reflex to a metronome has been formed, we apply the metronome over months without reinforcement, the reflex to it should surely have subsided long before, and hardly any trace of it should be left. So why is it that the character of this stimulus as a conditioned defensive one is somehow 'preserved' when it is accompanied by a movement constituting a conditioned inhibitor? One has the impression that this very movement somehow protects the stimulus against extinction, and thus the conditioned reflex second type is maintained perpetually. But we are unable to explain the causes of this phenomenon.

In conclusion to this section we would like to point out that

non-reinforcement of the defensive conditioned reflex second type by any perceptible unconditioned reflex has the effect that, once the conditioned reflex has been created, we cannot perform any 'operations' on it similar to those possible on conditioned reflexes of the first type, and on alimentary conditioned reflexes of the second type. Thus, we cannot extinguish it, or differentiate it, or delay it, nor can we form a conditioned inhibition to it, etc. Like the unconditioned reflex, this reflex is in a sense inviolable. Anything we wish to do with it has to be done before its formation, then we can differentiate two similar stimuli, reinforcing only one of them by the introduction of acid, we can form a delay inhibition, etc. All these plastic changes would be passed on to the conditioned reflex second type, but after its formation this is no longer possible.

8

Now we turn to a *fourth variety* of conditioned reflex second type, in which the movement, e g the flexion of the limb, is reinforced by a negative unconditioned stimulus. Then the animal learns to perform an antagonistic movement, namely extension The stimulus to this movement may be either a specially applied external stimulus, accompanying the flexion movement, or simply a skin stimulus, arising with a mechanical flexion of the limb In this latter case, to each attempt at passive flexion of the limb the dog will perform a straightening movement. It is clear that this reflex is distinguished from the third variety of conditioned reflex second type only in regard to its formation, and apart from this it possesses exactly the same properties as the other.

In summary of the foregoing, we give a table of all the four varieties of conditioned reflexes of the second type

Variety	Compound $s+r$	Stimulus s applied separately	Unconditioned reinforcing stimulus	Form of conditioned reflex second type
First	Reinforced	Non-reinforced	Positive	$s \dashrightarrow r$
Second	Non-reinforced	Reinforced	Positive	$s \dashrightarrow \sim r$
Third	Non-reinforced	Reinforced	Negative	$s \dashrightarrow r$
Fourth	Reinforced	Non-reinforced	Negative	$s \dashrightarrow \sim r$

s, external stimulus
r, movement applied, or corresponding proprioceptive compound stimulus
$\sim r$, movement antagonistic to r

9

As we have said again and again in the preceding sections, the effect of the conditioned reflex second type is never specifically bound up with the stimulus to which it has been formed, both in the field of alimentary reflexes, and in that of defensive reflexes there can be interchanges of responses of different reflexes in the same group (in the class of defensive reflexes, even between various groups of reflexes), and the provoking factors capable of eliciting the acquired movement are: for alimentary reflexes, stimuli causing a fall of excitation in the alimentary centre; for defensive reflexes, stimuli causing a rise of excitation in the corresponding unconditioned centre. The problem we shall now consider is, whether it is possible to have interchanges of responses between alimentary and defensive reflexes of the second type· i.e. can the dog use a movement elaborated as an alimentary one for the purpose of 'protection' against a negative stimulus, and, conversely, can it 'demand food' with the aid of a movement elaborated for defence?

This problem, which we have dealt with in a special work,* is complex, and we are not in a position to discuss it in detail here. We give briefly only the most important of the data we have obtained.

In our type of experiment (i.e. the dog at the stand, trained movements consisting in raising the limbs, the stimuli for these movements usually being indifferent stimuli), the relations are as follows:

(1) In general, between alimentary and defensive movements (as we shall, for simplicity's sake, call the responses of the respective conditioned reflexes of the second type) interchanges occur to a very weak extent. In other words, if both alimentary and defensive conditioned reflexes of the second type have been formed in the dog, then appropriate alimentary stimuli will provoke chiefly alimentary movements, whereas stimuli evoking a defensive reaction will provoke chiefly defensive movements.

(2) In experiments of this kind it is necessary to reckon with the circumstance that the alimentary or the defensive attitude

* Konorski, J, 'Zasady przełączania korowego' ('Principles of cortical switching'), *Przegląd Fiz Ruchu,* 1939, vol. ix, p. 191

of the dog is often marked by a certain inertia, which reveals itself in the following manner. If, after a series of alimentary conditioned stimuli, we apply a stimulus evoking normally a defensive movement, we shall find that the dog's immediate reaction to this stimulus is salivation and performance of the alimentary movement, and that only as the stimulus continues is the alimentary reaction replaced by the defensive reaction; the secretion of saliva stops and the defensive movement appears. The very next application of this stimulus gives a pure defensive reaction. Conversely, if after a series of conditioned defensive stimuli we apply an alimentary conditioned stimulus of the second type, we may find that the defensive movement appears at first; but experimental analysis will show that this is because this stimulus, acting after a series of defensive stimuli (i.e. against the background of a defensive attitude), has temporarily acquired defensive properties In different dogs this property of inertia is manifested to different degrees, and in some animals it is not to be observed at all. But it has to be taken into account when interpreting the facts.

(3) However, independently of this factor of the inertia of unconditioned states (which, by taking certain precautions, we can counteract) the interchange of alimentary and defensive movements does sometimes take place.

If we carry out extinction of an alimentary conditioned stimulus of the first type, then, as we have said, at first alimentary movements are performed in abundance, since this stimulus, as a fresh inhibitor, has become a provoking factor. Gradually, together with the extinction of the saliva reaction, the above movements also disappear However, if we continue to apply a non-reinforced conditioned stimulus, then we may observe that with very deep extinction (sometimes after several days of acute extinction) defensive movements begin to appear more or less regularly to this stimulus. We have the impression that these movements continue to retain their defensive character, and appear as a reaction against the application of the conditioned stimulus so many times without reinforcement, which stimulus to some extent acquires the properties of a negative stimulus.

The converse phenomenon, with alimentary movements appearing to the stimulus of a defensive reaction, also sometimes occurs, but the conditions governing its occurrence are not clear.

The facts above described show that among the determining factors designating which of the second type motor responses may appear in the given situation, the unconditioned background (alimentary or defensive) plays an important part. Stimuli evoking an excitation of the alimentary centre create a disposition for alimentary movements to be performed, but stimuli evoking excitation of the defensive centres create a disposition to defensive movements.

10

The factual material presented in the preceding sections allows us to draw certain conclusions concerning the *mechanism* of the formation and course of conditioned reflexes of the second type. Although we consider that the problem of this mechanism in all its extent is still far from solution and calls for much more experimental work, attempts at a solution can already be undertaken, and may at least serve as working hypotheses, providing material and stimuli to further experimentation.

Let us endeavour first and foremost to pick out those elements in the process of formation and course of conditioned reflexes second type which seem to be fundamental to the problem posed.

(1) Conditioned reflexes of the second type always arise against a background of certain situations, which in the case of alimentary reflexes are: non-reinforcement of the alimentary conditioned stimulus (of the first type) by food, or the withdrawal of food, and, in the case of defensive reflexes. the action of an excitatory conditioned stimulus, or a negative unconditioned stimulus. In other words, the animal learns to perform some movement when, despite its expectation, it does not get food, or when it is expecting the advent of an unconditioned negative stimulus. On the contrary, in situations in which the animal is expecting food, which it will *certainly* get, or when it is already receiving food, i.e. during the action of an alimentary conditioned reflex or an unconditioned reflex, a conditioned

reflex of the second type is never formed. Putting it briefly, the conditioned reflex of the second type is formed against the background of a fall of active excitation in the alimentary centre, or that of a rise of excitation in a defensive centre.

(2) If, against the background of the states described in point (1) above, some movement is performed which leads to the reception of food, or to the avoidance or elimination of the unconditioned negative stimulus (i.e. if this movement becomes an alimentary conditioned stimulus, or a conditioned defensive inhibitor), then it is established, i.e. it begins to be performed 'spontaneously', in the circumstances attendant on the foregoing procedure. If, on the contrary, a movement provoked in or performed by the animal is not reinforced by food, or is reinforced by an unconditioned negative stimulus (i.e. if it becomes an inhibitor of the alimentary reflex or a conditioned stimulus of the defensive reflex), then this movement is inhibited, this being accompanied by a movement antagonistic to it. So the given movement is established only when its performance averts a fall of excitation in the alimentary centre, or a further excitation, or rise of excitation in the defensive centres.

(3) The stimuli causing the performance of the established movement are the compounds of stimuli (extero- and interoceptive) which were witnesses of its formation and fixation, i.e. in the presence of which this movement played the part indicated in point (2) above. Stimuli in the presence of which the movement ceased to play this role (i.e. was not a means of receiving food or of avoiding a negative stimulus) begin to cause an inhibition of the movement, with an antagonistic movement making an accompanying appearance.

(4) A movement formed and fixed in the above-described manner is executed by the animal when the factors specified in point (1) come into action In other words, the animal performs the given trained motor reaction when there is a fall of excitation in the alimentary centre or a rise of excitation in the defensive centre On the other hand, active excitation of the alimentary centre, like inhibition of the defensive centre, never causes acquired movements to be performed.

Now to co-ordinate these facts into a general system.

Laboratory practice and everyday observation show that in the situations described in point (1) there is always a general motor excitation of the animal. Thus, both the withdrawal of the animal's food (or its insufficiency to satisfy the animal) and the application (best of all several times over) of the alimentary conditioned stimulus without reinforcement, or of a fresh differential inhibitor, always evoke a lesser or greater motor restlessness in the dog, such as shifting from paw to paw, vocal reactions, etc. A similar state of affairs is observable during the operation of an active conditioned or an unconditioned defensive stimulus. On this basis we are justified in assuming that the state we are discussing is due to some general excitation of the motor cortex, the nature of which has still to be investigated. Without going into an analysis of this state, we shall call it 'a state of exaltation' of the motor cortex.

Thus, both a fall of excitation in the alimentary centre, and a rise of excitation in defensive centres, cause a state of exaltation to arise in the motor cortex, which state, as we have shown above, is an indispensable condition of the formation and recurrence in dogs of conditioned reflexes of the second type.

If, with such a state of exaltation as the background, in the presence of a certain compound of stimuli a movement is provoked, then, between the cortical centres of these stimuli and the centre of the movement,* synaptic connexions arise. These connexions are either of an excitatory or of an inhibitory character, according to the role played by the movement itself. If its performance leads to a renewed rise of excitation in the alimentary centre or to a fall of excitation in the defensive centre, i.e. to an *abolition* of the state of exaltation in the motor cortex, then excitatory connexions arise between the corresponding centres. But if this movement does not lead to an abolition of the state of exaltation, or even incites this state, inhibitory connexions arise between the corresponding centres; then the accompanying

* When we speak of a 'centre of movement' we understand the term in a general sense, not attempting to be precise as to whether it is a centre of the proprioceptive stimuli corresponding to this movement, or the actual motor centre At present the structure of motor centres is so little known that it seems quite fruitless to speculate on the subject of their intimate mechanism.

stimuli evoke an inhibition of the provoked movement, bound up with elicitation of the antagonistic movement.

We would not wish to make any claim that the above explanation makes the phenomena under discussion completely intelligible, and does not require further development. But we consider that in this interpretation we have gone as far as the present state of our knowledge permits.

In order to achieve greater clarity, let us compare the mechanism of the formation of a conditioned reflex second type with that of the formation of conditioned reflexes first type.

We recall that in conditioned reflexes of the first type the synaptic connexions arose between the centre of the 'indifferent' stimulus and the unconditioned centre when they were concurrently excited, and that if the excitation of the first centre occurred during a rise of excitation in the second, the synaptic connexions then being formed had an excitatory character, if the excitation of the first centre occurred during a fall of excitation in the unconditioned centre, synaptic connexions of an inhibitory character arose. In conditioned reflexes of the second type the connexions arise between the centre of the indifferent stimulus and the *motor centre of the cerebral cortex*, the indispensable condition of conditioning being that the motor area must be brought into a 'state of exaltation'. This state causes the motor centres to acquire the temporary capacity to form synaptic connexions (directed towards these centres) with other centres coupled with them. In other words, the motor centres of the cortex temporarily take on a similarity to unconditioned centres (or to their cortical representations) which possess the above capacity more or less permanently.* So if in such a state a definite movement is provoked in some way or other, synaptic connexions are formed between the centres of the accompanying stimuli and the centre of the movement. These connexions have an excitatory character when the performance of this movement

* Permanence is, of course, relative The association of an indifferent stimulus with the introduction of food into the mouth of a completely satiated animal does not lead to the formation of an alimentary conditioned reflex, first type, just as the simple association of an indifferent stimulus with passive flexion of the limb does not lead to the formation of a conditioned reflex second type

abolishes or reduces the state of exaltation, but an inhibitory character when it maintains or increases that state. We are not yet in a position to explain why this happens, any more than we are able, for that matter, to say why excitatory or inhibitory connexions arise in conditioned reflexes of the first type according to the sequence in which the conditioned and unconditioned stimuli are applied.

It is easy to show that the interpretation we have just given of the formation of conditioned reflexes, second type, satisfactorily enables us to comprehend and systematize all the experimental material we have presented in this chapter. By way of example we shall analyse from this aspect the most important relevant facts.

There is no difficulty in elucidating the formation of the first variety of conditioned reflex second type. In § 3 above, on the basis of experimental evidence, we showed that this reflex is formed against a background of a fall of active excitation in the alimentary centre (when during consumption food is removed or expected food does not come), i.e against a background of a state of exaltation of the motor cortex. In this situation the provocation of a definite movement and its reinforcement by food leads to the formation of synaptic connexions between the centres of the indifferent stimuli which accompany the procedure and the given motor centre. These connexions are of an excitatory character, since the movement, becoming a signal of food, acquires the properties of an excitatory conditioned stimulus; consequently its centre sends excitatory impulses to the alimentary centre, and these, averting the fall of excitation in this centre, abolish the state of exaltation in the motor area.

But let us consider what happens when the movement thus formed ceases to be reinforced by food, i.e. when we carry out acute extinction of the conditioned reflex second type, the course of which is shown in fig 17.

In the initial phase of extinction active movements appear in great abundance in quick succession. This is understandable, since the dog, not receiving food, performs a movement which in the given circumstances provides the alimentary centre with excitatory impulses. However, this movement is not reinforced,

and in consequence the active excitation in the alimentary centre falls again, which causes an immediate reappearance of this movement. This process continues so long as the given movement is an active conditioned stimulus, i e. so long as it provides excitatory impulses to the alimentary centre. Gradually, however, its non-reinforcement results in inhibitory connexions being formed in greater and greater quantities between the centre of the proprioceptive stimulus and the alimentary centre, and in consequence the active excitation of the alimentary centre evoked by the performance of this movement grows weaker and weaker. Therefore the movement ceases to avert a fall of the excitation in the alimentary centre, i.e. it no longer abolishes the state of exaltation of the motor cortex, and in consequence inhibitory connexions arise between the centres of the accompanying stimuli and the centre of this movement. And so the motor reaction gradually subsides, and antagonistic motor reaction arises in its place If this reaction consists in an extension of the limb it is of course difficult to perceive, but if the trained movement is that of extension, and the antagonistic movement is flexion, the latter is quite perceptible (cf. p. 227).

An antagonistic movement appearing in the extinction of a conditioned reflex second type soon subsides if it is not reinforced. But if we cultivate and reinforce it (as is the case in the second variety of conditioned reflexes second type), this movement becomes an alimentary conditioned stimulus (first type); as such it begins to oppose the fall of excitation in the alimentary centre, and in consequence, in the circumstances in which it was evoked, it becomes fixed and is performed regularly.

Similarly, we can analyse and explain the mechanism of the formation of defensive reflexes second type (third and fourth varieties) In the third variety a movement is provoked in a state of exaltation of the motor cortex produced as the result of the action of an excitatory defensive conditioned stimulus. The movement is followed by the withdrawal of this stimulus without successive application of the unconditioned stimulus, i.e. the state of exaltation is abolished. So it is clear that excitatory connexions arise between the centres of accompanying indifferent stimuli and the centre of the movement, and in

consequence the movement begins to be performed in the circumstances in which it was provoked. But if, when provoked in these circumstances, it is reinforced by an unconditioned negative stimulus, i.e. it itself becomes a conditioned stimulus of a defensive reaction, its performance not only fails to assuage the state of exaltation of the motor cortex, but, on the contrary, enhances this state, and in consequence inhibitory connexions arise between the centres of the accompanying stimuli and the centre of the movement. Then we get the fourth variety of the conditioned reflex second type.

Similarly, there is no difficulty in clarifying the phenomena of cortical switching, which we discussed in §§ 5 and 9. We recall that the performance by the animal of a definite movement was found to be dependent on the one hand on determining factors, which designate what movement is to be performed, and on the other on provoking factors, which give the 'executive order'. It is clear that the determining factors are those whose centres have been associated with the centre of the given movement by excitatory connexions, while the provoking factors are those which cause a state of exaltation in the motor cortex and somehow provide impetus to the execution of one of the formed movements.

Here the important question arises, to what extent is a state of exaltation of the motor cortex necessary to the execution of the movement when it is adequately formed and fixed? We are unable to give a final answer to this question. So far as defensive reflexes are concerned it seems highly probable that the movement (with the aid of which the animal prevents the application of a negative stimulus) is performed only when a corresponding 'threat' arises, i.e. when signals heralding a negative stimulus, and therefore causing a state of exaltation of the motor cortex, come into being. But so far as alimentary reflexes are concerned, the position is by no means so clear. In fact, when a movement regularly used in feeding (e.g. in the case of a monkey, the hand movement of seizing food and raising it to the mouth) is firmly established it becomes an integral part of the act of feeding, and it is difficult to assume that the impossibility of direct reception of food, i.e. a state of exaltation of the motor cortex, is always

an indispensable condition of its performance. In experiments with monkeys we sometimes noticed* that an animal which *could* obtain the food directly none the less performed a certain acquired and firmly established movement, in a 'ritual' manner, so to speak, though it was completely unnecessary. Similarly, we frequently observed that a monkey performed a conditioned motor reaction of the second type during the actual act of eating, and therefore during an unconditioned stimulation of the alimentary centre (never, however, observed in a dog). In these conditions it is difficult to assume a state of exaltation in the motor cortex. Moreover, so far as dogs are concerned, we were able to observe that a completely satiated animal executed the trained movement to the given stimulus, even though it did not eat the food which, thanks to this movement, was offered to it. So we have to ask whether the performance of the movement was the result of alimentary excitation, or whether the dog executed the movement only because of prolonged and intensive training.

Obviously the problem calls for further study, and of course the degree of phylogenetic development of the animal must be taken into account.

11

In preceding sections it was shown that conditioned reflexes of the second type form a specific kind of plastic changes with a mechanism differing essentially from the plastic changes which constitute conditioned reflexes of the first type. But so far as *established* conditioned reflexes of the second type are concerned, it is easy to demonstrate that in respect of their excitability properties they are very similar to conditioned reflexes of the first type, and perhaps are even identical with them. Although we do not yet possess sufficient experimental evidence to prove this completely, the data we have collected are largely in its favour.

For instance, we have been able again and again to observe that the principles of both external inhibition of active conditioned reflexes and the disinhibition of inhibitory conditioned reflexes by extraneous agents wholly apply to conditioned

* Konorski, J. and Norkina, L., unpublished experiments

reflexes of the second type. Similarly, we have established that the strength of the motor reaction (i.e. the magnitude of the movement, its rapidity and its latency) to a large extent depends on the excitability in the centre of the reinforcing unconditioned stimulus. For instance, when the animal is satiated the alimentary motor response of the second type possesses an extraordinarily long latency, and sometimes occurs very slowly, in contradistinction to the energy and rapidity with which this same movement is executed when the animal is hungry. Similar relations also occur in defensive reflexes of the second type. Finally, we have obtained clear evidence that in both defensive and alimentary reflexes of the second type the law of the strength of conditioned stimuli is binding, i.e the strength of the reflex depends on the strength of the conditioned stimulus. Weak conditioned stimuli usually produce a weaker, slower motor reaction, with a longer latency, than strong stimuli. In certain conditions (e.g. when the animal is satiated) these differences emerge with extraordinary clarity, so enabling us to define this state as a typical 'narcotic phase'.

Obviously it would be very interesting to make a more detailed investigation of the other excitability properties of the conditioned reflexes of the second type in regard to summation, induction, etc. But we think that the evidence we possess allows us to express the supposition that while the mechanism of the formation of different plastic changes may be very various, the mechanisms of the *course* of conditioned reflexes are identical, and completely accord with the course of innate reflexes.

12

It would carry us too far from our main object to compare and confront our conception of the formation of conditioned reflexes of the second type with the conceptions of various American psychologists, such as Thorndike, Guthrie, Hull, Skinner, and others.* We have engaged in an extensive discussion and

* Thorndike, E. L , *Animal Intelligence*, New York, 1911; *Fundamentals of Learning*, New York, 1932; *Psychol. Rev.* 1938, vol XLV, p. 191, *et alia*. Guthrie, E. R , *The Psychology of Learning*, New York, 1935 Hull, C. L , *Psychol. Rev.* 1930, vol. XXXVII, p. 241; ibid. 1930, vol. XXXVII, p. 511;

criticism of their views elsewhere.* Here we only point out in passing that, as is easy to see, there are certain elements in common between our conception and those of the above-mentioned authors (especially Hull and Guthrie). However, the differences are no less important. They concern both the method of approach, which in the case of these writers is more descriptive, and the deductions drawn from analysis of the problems discussed. This is not surprising, because in order to construct their theories the American writers made use of facts often taken from every day observation or from experiments far too complicated for elementary analysis (e g. an animal's run through a maze, behaviour in an experimental box, etc.), whereas our experiments were constructed in as simple a manner as possible, and their *object* was to elucidate the mechanism of the phenomena represented in them.

None the less it is not difficult to show that, with the experimental evidence at our disposal, we can easily and with great precision analyse and explain a number of the phenomena of animal behaviour provided by behaviourist psychology.

In order to demonstrate this, let us analyse the behaviour of an animal, based on the so-called method of trial and error, such as in the opening of an experimental box and in maze-habit.

Let us assume that through the bars of the cage the animal sees food. The sight of it is, of course, a powerful 'natural' conditioned stimulus, i.e. it evokes a strong excitation in the alimentary centre. But as the food cannot be reached, there follows an abrupt inhibition of the alimentary reflex (so-called 'unceasing extinction') which entails a fall of active excitation in the alimentary centre (if we examined the secretion of saliva, we would be able to measure its extent). This causes a state of exaltation of the motor cortex, and consequently the animal begins to execute the movements in its repertory, the determining factor being the given experimental situation. So it will

ibid 1932, vol XXXIX, p 25, ibid 1937, vol XLIV, p. 1 Skinner, B. F., *J. Gen. Psychol.* 1932, vol. VII, p. 274, ibid, 1935, vol. XII, p. 66; ibid. 1937, vol XVI, p 272.

* Konorski, J., *Usp. Sovrem. Biol.* 1942, vol. XV, p. 4.

not leap up, or scrape the ground, as it would if the food were hung up or buried, but will gnaw at the bars, will try to bend them aside, will stretch out its paw towards the food, etc. These movements, which are ineffective, gradually subside; although the animal returns to them from time to time as the result of a regression in the extinction, this happens more and more rarely. But the movement leading to acquisition of the food, being reinforced, has a tendency in the given situation to become fixed.

The maze habit is a chain conditioned reflex of the second type which we have succeeded in completely reproducing with the aid of conditioned reflex experiments.* If a stimulus is a conditioned alimentary one of the first or second type, it obviously can serve as a reinforcing agent for another conditioned reflex, as it does in conditioned reflexes of the second order. Thus we can create a number of conditioned reflexes of the second type, of which each preceding reflex is reinforced by the conditioned stimulus of the following reflex, according to the pattern:

$$s_1 \rightarrow r_1 — s_2 \rightarrow r_2 — s_3 \rightarrow r_3 \ldots s_n \rightarrow r_n — S_u^+$$

($\rightarrow$ means evokes, — entails, $s_1, s_2, s_3, \ldots s_n$ are conditioned stimuli, $r_1, r_2, r_3 \ldots r_n$ are various motor responses, S_u^+ the presentation of food.)

But as each of the following movements, being closer to the unconditioned stimulus, is a stronger conditioned stimulus and, as such, has a greater tendency to be performed, consequently in chain reflexes we very often observe 'anticipation', consisting in the given movement appearing earlier than it should, and only its non-reinforcement leads to the performance of the 'correct' movement. So the formation of a chain reflex is usually based on a special kind of differentiation, when to the earlier stimulus the animal must inhibit a later reaction, which enables it to execute the proper reaction to this stimulus. As for extinction of a chain reflex, it begins, of course, from the end, since those elements of the reflex which are nearest to the unconditioned stimulus are most strongly inhibited.

* Konorski, J and Miller, S., *Podstawy fizjologicznej teorii ruchów nabytych* (*Bases of the Physiological Theory of Acquired Movements*), Warsaw, 1933, p. 99; Konorski, J. and Norkina, L , unpublished experiments.

It must be pointed out that Hull has provided a similar analysis of the experiments we have described. The difficulty which he came up against was due to his attempting (in our view ineffectively) to force the phenomena he analysed into the scheme of conditioned reflexes of the first type.

In conclusion we must point out that it would be erroneous to consider that conditioned reflexes of the first and second type completely exhaust the acquired behaviour of higher animals and man. One can easily demonstrate that there are other forms of behaviour, which have not so far been subjected to physiological analysis, as for instance, various kinds of intelligent behaviour investigated by psychologists. The experiments of W. Kohler and Norman Maier* seem to be especially instructive in this regard. It seems to us that by way of an exact analysis of these experiments and separation out of their essential elements, it would be possible to construct appropriate conditioned reflex experiments, which would illustrate the corresponding facts (just as we have done in the case of conditioned reflexes of the second type) and thus to set about their physiological study. Before the outbreak of war in 1939 we had begun work in this direction on dogs, monkeys and children. Unfortunately it had to be broken off, though its first results seemed to be highly promising.

* Kohler, W , *The Mentality of Apes*, New York, 1925, Maier, N R F , 'Reasoning in white rats', *Comp Psychol Monogr* 1929, vol VI, no 29

CHAPTER XIII

Nomenclature of the physiology of higher nervous activity

The reorientation of the physiology of higher nervous activity which we have attempted in this work necessitates extensive changes in nomenclature.

To enable the reader to follow the changes that need to be made, we give a table (p. 251) of the main features of higher nervous activity discussed in this book. The following is a brief commentary to this table.

In the first column are given the *descriptive* names or, where names are lacking, a brief description of the corresponding phenomena. The second column gives the names arising from the Pavlovian interpretation of the mechanism of the phenomena, or the interpretation itself. In the third column are the names it is proposed to adopt for these phenomena in accordance with their analysis in the present work, or a description of their mechanisms. For instance, external inhibition is a descriptive name serving to denote the phenomenon consisting in a fall in the magnitude of the conditioned reflex under the effect of extraneous stimuli. This name involves no conclusion as to the mechanism of the phenomenon. Negative induction is the name given to this phenomenon in accordance with the Pavlovian conception of cerebral activity. This name connotes that excitation of the centre of the extraneous stimulus produces inhibition around this centre which explains the reduction in the conditioned reflex. Inhibition of the conditioned reflex under the influence of an antagonistic reflex (interference between reflexes) is the class to which we have allocated this phenomenon on the basis of our analysis.

Items 1 to 8 concern the properties of active conditioned reflexes (their formation, generalization, external inhibition, top value and interaction between homogeneous conditioned reflexes). They were discussed in detail in Chapters VI—VIII. Items 9–16 are devoted to internal inhibition (i.e. its formation, inhibitory

after-effect, disinhibition and induction), discussed in detail in Chapters IX and X. Finally, items 17–19 concern problems which were only touched on in passing in this book, namely, the so-called hypnotic phases and the phenomenon of sleep. The fourth column of the table indicates the chapters and sections in which the various problems are discussed

So far as the first column is concerned—the descriptive names—there is no reason whatever why any changes should be made. These names are in the main convenient and have been well chosen, moreover they have been generally adopted (especially in psychology) so that changes would only cause confusion and misunderstanding. And so we think they should all remain unaltered.

But when we turn to the second column, from all we have said it is obvious that these terms must be rejected in their entirety. The names in the first column do not convey any suggestion as to the mechanism of the phenomena in question, but those in the second group say a great deal, and what they say is inaccurate. To leave them in use would only cause confusion.

There are hardly any new names in the third column. The great majority are taken from the general physiology of the central nervous system, mainly from the vocabulary of the Sherrington school, which has been completely adopted in current scientific usage. Certain of them have been modified a little to adapt them to the specific properties of higher nervous activity.

Thus, so far as the terminology of physiology of the higher nervous activity is concerned we completely reject the terms: irradiation and concentration of excitation and inhibition, positive and negative induction, the top capability of cortical cells, and top inhibition. As can be seen from the table, the phenomena which were given these names have been regrouped and explained in a quite different manner. As we said in Chapter X (§ 2) we have decided to leave the name 'induction' for the phenomena called 'positive induction' in the Pavlov system, because this term approximately coincides with the Sherrington term 'immediate induction'. But the meaning we

give the term is quite different from that of the Pavlov theory. For in this theory it denoted the rise of excitability of the nerve cells surrounding the focus of inhibition (or the rise of excitability of the cells after their inhibition), whereas we take it as meaning the summation of the excitatory and inhibitory conditioned reflex, resulting in a more or less strong facilitation. Therefore disinhibition of an inhibitory reflex preceded by an excitatory conditioned reflex is according to our terminology a typical manifestation of induction, whereas in the Pavlov scheme this phenomenon was allocated to a completely different category.

As we have indicated above, the term 'top of capability of the cortical cells' must be completely dropped. However, in its place we have decided to introduce the purely descriptive term 'top value of conditioned reflexes', or 'top of conditioned reflexes', to denote the greatest magnitude of these reflexes achievable in a given animal, in given conditions with given reinforcement.

As for the phenomena corresponding to the so-called 'hypnotic phases', we have used 'equalizing relations between reflexes', and 'paradoxical relations between reflexes' as descriptive terms. These describe the phenomena to which they refer, without suggesting any explanation. We have seen that these phenomena have to be explained, at least in certain cases, by reference to antagonistic inhibition caused by a defensive reflex. Whether the term 'hypnotic phases' is theoretically valid for other cases can only be decided by future research.

Comparison of the various columns and items of the table reveals the fundamental changes that have been made in the systematization of phenomena in the field of science under discussion.

On the one hand certain phenomena to which the Pavlov system attributed one and the same mechanism have been separated. For instance, according to the Pavlov theory the generalization of conditioned reflexes (item 3), the disinhibition of inhibitors by an active conditioned stimulus (item 15) and by an extraneous stimulus (item 16) have a common mechanism—the irradiation of excitation. In our view each of these pheno-

mena has a quite separate mechanism · generalization depends on the partial overlapping of the conditioned centres, disinhibition of the inhibitory reflex by the active-conditioned reflex is an expression of summation and facilitation, disinhibition by an external stimulus is probably due chiefly to interference between heterogeneous reflexes. Similarly, in the Pavlov theory, inhibitory after-effect (item 11), the direct inhibitory influence of a strong inhibitory stimulus on active conditioned reflexes (item 13), and sleep (item 19), are all manifestations of the irradiation of inhibition. In our conception the first of these is the result either of generalization of the inhibitory stimulus, or of the inertia of inhibition in the unconditioned centre; the second is the result of the predominance of the inhibiting influence of the inhibitory reflex over the facilitating influence; the third is a quite distinct phenomenon, with a sub-cortical mechanism. Finally, according to the Pavlov theory external inhibition, being an expression of negative induction (item 4), and increase of the conditioned reflex after-current to the inhibitory reflex, being an expression of positive induction (item 12), are symmetrical phenomena. In our view they have nothing whatever in common.

On the other hand, certain phenomena which Pavlov assumed to possess quite separate mechanisms we have explained by reference to one mechanism. For instance, according to Pavlov, increase of the active conditioned reflex after-current to the inhibitory reflex (item 12) and disinhibition of the inhibitory reflexes after-current to the active conditioned reflexes (item 15) are the expression of two completely different mechanisms, the first of positive induction, the second of the irradiation of excitation. In our conception these two phenomena have an identical mechanism, and are both the result of summation and facilitation of supraliminal and subliminal conditioned reflexes. Similarly, external inhibition of conditioned reflexes (item 4) is explained according to Pavlov by reference to negative induction, and the diminution in the conditioned reflexes to supra-maximal stimuli (item 6) by reference to top inhibition. In our conception these two phenomena have exactly the same mechanism, being caused by antagonistic inhibition, evoked by a defensive reflex.

The difference between them consists solely in the circumstance that in the first case the alimentary conditioned reflex and the defensive reflex are evoked by two different stimuli, whereas in the second case the source of both the reflexes is one and the same stimulus.

All of which goes to show how extensively the whole field of knowledge under consideration has been resurveyed, and how thoroughly it has been reorganized.

No.	Descriptive term or description of the phenomenon	Mechanism or name of the phenomenon according to Pavlov's theory	Mechanism or name of the phenomenon according to the new conception	Chapter and Section reference
1	Excitatory or active conditioned reflex	Excitation of the conditioned centre as the chief feature of the phenomenon	Excitation of the unconditioned centre as the chief feature of the phenomenon	Ch. VI
2	The formation of the conditioned reflex	'The meeting of the waves irradiated from different points' (Pavlov)	The formation of excitatory synaptic connexions between two coupled centres as the result of their concurrent excitation	Ch. V, § 4 Ch. VI, § 2
3	Generalization of the excitatory conditioned reflex	The irradiation of excitation from the centre of the conditioned stimulus	Partial over-lapping of the centres of the primary and secondary conditioned stimuli	Ch. VIII
4	External inhibition	Negative induction	Inhibition of the conditioned reflex by an antagonistic reflex (interference between reflexes)	Ch. VII, § 2
5	Top value of conditioned reflexes	Top capability of cortical cells	The 'state of saturation' of the connexions between the conditioned and unconditioned centre; occlusion	Ch. VI, § 2

No.	Descriptive term or description of the phenomenon	Mechanism or name of the phenomenon according to Pavlov's theory	Mechanism or name of the phenomenon according to the new conception	Chapter and Section reference
6	The diminution of the conditioned reflex to the supramaximal stimulus	Top inhibition	The inhibition of the conditioned reflex by an antagonistic defensive reflex (interference)	Ch. VII, § 3
7	Interaction of homogeneous conditioned reflexes to weak stimuli	Summation either in conditioned centres or in unconditioned centre (?)	Summation and facilitation in unconditioned centre	Ch. VII, § 1
8	Interaction of homogeneous conditioned reflexes to strong stimuli	Top capability of cortical cells	Occlusion in unconditioned centre	Ch. VII, § 1
9	Internal inhibition (inhibitory conditioned reflex) (*a*) Experimental extinction (*b*) Delay (*c*) Differentiation (*d*) Conditioned inhibition	Inhibitory process occurring in the conditioned centre	Inhibition and excitation of unconditioned centre (*a* *b*, *c*) Pure inhibition of unconditioned centre (*d*)	Ch. IX
10	The formation of the inhibitory conditioned reflex	The establishment of 'inhibitory excitability' in the conditioned centre	The formation of inhibitory connexions between two centres as the result of coincidence in excitation of conditioned centre with fall of excitation in the unconditioned centre	Ch. IX, § 1
11	Inhibitory after-effect	Irradiation of inhibition from the centre of the conditioned stimulus	(1) Generalization of the inhibitory reflex (2) Inertia of inhibition in the unconditioned centre	Ch. X, §§ 4, 5

No.	Descriptive term or description of the phenomenon	Mechanism or name of the phenomenon according to Pavlov's theory	Mechanism or name of the phenomenon according to the new conception	Chapter and Section reference
12	The increase of the excitatory conditioned reflex concurrent with the inhibitory reflex	Positive induction between the centres of inhibitory and excitatory conditioned stimuli	Summation of the excitatory conditioned reflex and the excitato-inhibitory conditioned reflex with facilitation predominating (immediate induction)	Ch x, § 2
13	The decrease of the excitatory conditioned reflex concurrent with the inhibitory reflex	Irradiation of inhibition from the inhibitory conditioned centre	Summation of the excitatory conditioned reflex and the inhibitory (or excitato-inhibitory) conditioned reflex, with inhibition predominating	Ch x, § 3
14	Mutual disinhibition of inhibitory conditioned reflexes	Positive induction	Summation of two excitato-inhibitory conditioned reflexes with facilitation predominating (immediate induction)	Ch. x, § 2
15	Disinhibition of inhibitory conditioned reflex after-current to the excitatory conditioned reflex	Irradiation of excitation and 'washing out' of inhibitory excitability from the inhibitory conditioned centres	Summation of the excitatory conditioned reflex and the excitato-inhibitory conditioned reflex with facilitation predominating (immediate induction)	Ch. x, § 2
16	Disinhibition of the inhibitory conditioned reflex by extraneous stimuli	Irradiation of excitation from the centre of the extraneous stimulus	(1) Inhibition of the inhibitory reflex by antagonistic reflex (?) (2) Summation and facilitation of allied reflexes	Ch. x, § 2

No.	Descriptive term or description of the phenomenon	Mechanism or name of the phenomenon according to Pavlov's theory	Mechanism or name of the phenomenon according to the new conception	Chapter and Section reference
17	(*a*) The equalizing relations between conditioned reflexes (*b*) The paradoxical relations between conditioned reflexes	The lowering of the top capability of the cortical cells (equalization and paradoxical phase)	(1) Inhibition of the conditioned alimentary reflex by the defensive reflex (2) 'The indirect inhibition' (?)	Ch xi, § 3
18	The diminution of conditioned responses particularly to weak stimuli	Narcotic phase	The fall of excitability in the unconditioned centre	Ch. vi, § 3 Ch. xi, § 3
19	Sleep	The irradiation of inhibition over the cortex and subcortical ganglia	A subcortical phenomenon related to the sleep centre (?)	Ch. x, addendum

Chapter XIV

Conclusion

Looking back over the course of the work we have traced in the foregoing chapters, we think we have succeeded in demonstrating that reconstruction of the physiology of higher nervous activity, and its harmonization with the general physiology of the nervous system, are both necessary and possible. Here we briefly resume the essential points on which our attempt to achieve such a reconstruction has been based.

All our conception of higher nervous activity has been consistently founded on the neuronic theory of the functioning of the nervous system. All the ideas we have developed in this book fit perfectly into the framework of that theory, and we have advanced nothing in conflict with it. But it needs to be enlarged a little if it is to serve as a basis for understanding the activity of the cerebral cortex, i.e. the activity of an organ endowed not only with excitability but with plastic properties, an organ which not only transmits nervous impulses, or inhibits their transmission, but also creates new paths for them to travel along. The additional assumptions required in order to account for these properties are as follows:

Firstly, we have assumed that the elaboration of a conditioned reflex consists in the formation of new functional connexions in the brain, i.e. in a formation and multiplication of *synapses* between the concurrently excited coupled cerebral centres. Secondly, we have postulated that if a combination of excitations which results in a plastic change is not repeated within a certain period, a greater or lesser regression of this change occurs, in other words, the corresponding synaptic connexions undergo atrophy. Thirdly, we have assumed that internal inhibition, i.e. the process that arises with non-reinforcement of the established conditioned reflex, consists in the formation of new synaptic connexions of an inhibitory character, independently of the already existing excitatory synaptic connexions.

It seems to us that if the foregoing assumptions are added to

the general laws established for the central nervous activity, the main data in the field of physiology of higher nervous activity can be satisfactorily explained, and the lines and perspectives of the further development of this science can be projected. Then the physiology of higher nervous activity becomes a *genuine* branch of neurophysiology, entirely founded on the general laws of this science and drawing on its achievements. And so we dare to suppose that the work we have undertaken will not have been altogether useless, even if one or another of the various statements and formulations we have put forward should prove to be inaccurate in the light of future experimental work, and so to require correction.

In conclusion, we would like to state our own view of the scope and the limitations of our work, and of the importance it may possess for physiology of the nervous system

We begin by pointing out its limitations.

It is very important to realize that the problems which this book discusses concern only the *lowest levels of cortical co-ordination*, and it would be highly detrimental if, armed with the principles we have established, we attempted to subject to their domination all the phenomena of cortical activity in animals and men.

Even in the field of purely Pavlovian conditioned reflexes the investigator is continually coming up against facts which, because of their complexity, are not fully covered by the principles laid down in this book, and which necessitate the discovery of new ways for their complete understanding. Some examples of these more complex phenomena are the dynamic stereotype, which we discussed in Chapter XI, the orientation reaction to new patterns and combinations of stimuli, the conditioned reflex 'to time', the differentiation of complex compounds of stimuli, and so on. Although all these are facts that have been discovered in physiological experimentation, hitherto they have not been susceptible of strict physiological analysis, and they can be studied only externally, by way of pure description. In other words, in these cases we are held up at the level of investigations proper to behaviouristic research, which is concerned with precisely this kind of exact description and systematization of

the manifestations of cortical activity, but not with their deep physiological analysis.

Still further facts hitherto unsubjected to physiological analysis are to be found in the field of animal-motor activity, of which one of the simplest mechanisms has been isolated in the form of conditioned reflexes second type, described by us in Chapter XII. In that chapter we have drawn attention to the phenomena of so-called intelligent behaviour, and have stressed their distinctive character. In view of the present state of our physiological knowledge we think it perfectly reasonable that contemporary behaviourist psychology is engaged on the description and systematization of animal and human motor behaviour, without waiting for a corresponding development in physiological investigation.

But the phenomena least accessible to the physiological approach in the present state of our knowledge are those, of course, associated with human mental activity. It is true that many phenomena in this field are already susceptible of physiological analysis, and that such analysis contributes to their more profound and full understanding. But we would be falling into a serious error if we considered, as some do, that these latter phenomena exhaust all our cortical activity, and that in consequence the whole of the human mental processes can now be physiologically explained. It seems to us that the physiology of higher nervous activity is now approximately at that stage which physics reached several centuries ago, when Newton's genius laid the foundations of its modern development. Although by comparison with the present position the range of facts then known and comprehensible to physics was insignificant, it gained a stable basis and ground-work for further progress. And just as thereafter physics advanced continually for several centuries, conquering more and more phenomena of inorganic nature, and more and more profoundly elucidating the mechanisms of those phenomena, so the science of higher nervous activity, which by Pavlov's genius has been set on the firm ground of biological investigation, now has all the requisites for the mastery of ever widening fields of mental activity and their increasingly deep analysis.

But while there are phenomena of cortical activity not yet susceptible of an adequate physiological approach to them, there are others in which physiological analysis can already be begun. Among the important fields falling within this category is the pathophysiology of higher nervous activity.

We have deliberately excluded this field from our scope, since its study presents a new and considerable task, one all the more difficult to undertake because, we think, even the rich agglomeration of facts gathered in this field by the Pavlov school is not sufficient to enable us to achieve a synthesis. For, whereas in the investigation of the normal activity of the cerebral cortex we have been able to make use of ready-made schemes drawn from the physiology of the lower nervous activity, no such schemes are available in this field. And although the Pavlov school has established a great mass of facts, as they have been assembled under the guidance of fallacious principles, they cannot be satisfactorily adapted to the formulation of a new scheme. Indeed, as one reads the extremely interesting and factually rich works done by the Pavlov school on the subject of experimental neuroses, their aetiology, symptoms, and treatment, one cannot but feel that, although these works have contributed a great deal of positive value to our knowledge of the pathophysiology of the nervous system, they still remain in the category of raw material, since the interpretations given to these phenomena are often arbitrary and inexact. However, we believe that the task we have indicated is perfectly possible of realization, and, furthermore, that it is urgent, since its accomplishment will provide the vitally important pathophysiological bases for the study of nervous and mental disorders.

We consider that the inclusion of the physiology of higher nervous activity in neurophysiology, and its transformation into a normal branch of this science will not only open new horizons for itself, but also may contribute considerably to the enrichment of neurophysiology as a whole, by providing new conceptions and new methods. Again and again in the foregoing pages we have pointed out that, though the conditioned is distinguished from the ordinary reflex both by the manner of its establishment and by the manner of its maintenance, as an excitability pheno-

menon it possesses the same properties as the ordinary reflex. So these properties in conditioned reflexes can be investigated in exactly the same way as in ordinary reflexes, and the laws discovered in the course of such investigations will have validity for nervous activity as a whole.

Although it is only to be expected that investigation into conditioned reflexes will, perhaps, never achieve that degree of precision which has been gained in the study of lower nervous activity, none the less these reflexes possess certain properties which enable the physiologist to make a deeper analysis of certain aspects of nervous phenomena than is possible in the case of investigation into ordinary reflexes.

When dealing with the reflex preparation of a decapitated or spinal animal, we come up against a ready-made system of relations, defined and fixed in advance. It is true, of course, that out of the extremely rich central arrangements of the nervous system the investigator can at will select either two-, three-, or multi-neuron reflexes, whether active or inhibitory But the range of these reflexes is fixed and defined by the animal's own phylogenetical development. In the case of conditioned reflexes the situation is quite different. Here the conditions of the experiment are imposed by the experimenter himself, he himself establishes the reflexes he requires, he dictates his demands to nature, and is not compelled to subordinate his experiments to established patterns. Thus, in accordance with the requirements of his experiment he can elaborate a series of allied reflexes, evoked by the stimuli of one or of different analysers, of identical or different strengths; he can establish inhibitory reflexes of different depths and with different proportions of the processes of excitation and inhibition, he can *change* the intensity of this or that process, in accordance with his own desires. It is obvious that ordinary reflexes cannot provide such extensive possibilities, or allow such freedom in the utilization of the experimental object. And so we expect that in the course of further study of the relations between both allied and antagonistic, both excitatory and inhibitory reflexes, the method of the conditioned reflex should yield better and richer results than are gained by the present methods of investigation of spinal reflexes.

But whereas for the general physiology of the central nervous system the reconstruction of the field we have been discussing will be no more than an enrichment and supplementation of its achievements, for the science of higher nervous activity itself it is of fundamental importance, and the sooner it is carried through, the more rapidly will this branch of physiology emerge from the deadlock in which it is at present. For so long as the great edifice of knowledge built up by Pavlov was guided and extended under the direction of that great scientist's genius, it could continue to grow despite its isolation, and despite the fundamental inadequacy of its construction. But since Pavlov's death this science, rent as it is with intrinsic contradictions, submerged beneath a great mass of inexact and arbitrary interpretations of the facts, isolated from cognate fields, and with its own specific system of terminology and concepts which is wholly foreign to other branches of neurophysiology, has been unable to make any further progress. And unless a fundamental readjustment, such as we have attempted to make in the foregoing pages, is not made in its basic concepts and standpoint, the physiology of higher nervous activity is bound to suffer degradation in status, and Pavlov's priceless heritage will be largely wasted. It is at least a good sign that many investigators in this field, both inside and outside the Pavlov school, are more and more realizing the necessity for such a readjustment.

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